SEPTEMBER,
1939

The JOURNAL of SPEECH DISORDERS

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Official Publication, of the American Speech Correction Association in its 14th year.
Editorial Office: Speech Clinic, Ohio State University, Columbus, Ohio.
Volume 4 : Number 3

UNIVERSITY OF MICHIGAN
THE HORACE H. RACKHAM
SCHOOL OF GRADUATE STUDIES
ANN ARBOR
INSTITUTE FOR HUMAN ADJUSTMENT

$3.00 per Year
Single issue $1.00

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The JOURNAL OF SPEECH DISORDERS is published quarterly in March, June, September, and December of each year by the American Speech Correction Association; $1.00 a copy; $3.00 a year (Canada and foreign, $3.50). All communications and subscriptions should be sent to the editorial and business office: Speech Clinic, 324 Derby Hall, Ohio State University, Columbus, Ohio.

Entered as second-class matter at the Post-Office in Columbus. Ohio, under Act of Congress, of March 3, 1879.

THE SPEECH SURVEY*

D. W. MORRIS, Ph.D.
Indiana State Teachers College,
Terre Haute, Indiana

It is the purpose of this paper to review briefly some of the more important speech surveys, and to give summarized reports of two surveys of the author. There are two general types of speech survey. One type of survey is designed to contribute to the general knowledge of the number and kind of speech defects and serve as a general guide for the approach to a particular situation. Ordinarily in such a survey, a questionnaire method is used and the basis of judgment is usually the opinion of school teachers or school principals. The early surveys were of this type. In his report to the Board of Education of St. Louis in 1916, J. E. W. Wallin (7) reported an extensive survey of his own and summarized eight other surveys. Wallin found that 2.8% of the 89,057 children in the elementary and high schools of St. Louis had defects of speech. The prior surveys quoted by him showed the total number of speech defectives to range from 2.2% in the cases of a study in Denmark and another in New Orleans to 11.5% in the survey of the speech of 14,235 children by Rouma of Belgium. In these reports the incidence of stuttering is fairly constant, ranging from .61% to 1.4%.

Another type of survey is designed to serve as a specific guide for the approach to a particular situation. The investigation is ordinarily carried on by means of a personal interview with each member of a school population. A consideration of some of the surveys of this type will show that there is a tremendous variation in the incidence of speech disorders from time to time and from place to place, or else there is much disagreement as to what constitutes a speech defect. In 1916 Smiley Blanton (2) conducted a personal survey, "assisted by Erminie Ballard and Margaret Gray Blanton, of all the children, below the high school grade in the public and parochial schools of Madison, Wisconsin," a total of 4862 children, with an age range of four to eighteen years. This investigation reported the number of speech defects of all kinds at 5.9%, and showed a gradual decrease from the lower grades to the higher. "It is seen that the number of defects is greatest in the first grade and gradually diminishes until when the eighth grade is reached, they have practically disappeared." Five years later Dr. Blanton reported a survey of the speech of the freshman class at the University of

*A paper presented at a joint meeting of the American Speech Correction Association and the National Association of Teachers of Speech, December, 1938.

Wisconsin (1). He examined personally 1400 of the class of 2,240. He reported that 18.13% had defective speech, "were found to be unable to meet the necessities of English speech." It appears that the freshmen of the University of Wisconsin in the year reported were much less favored by fortune than the children of Madison public and parochial schools of 1916; or else Dr. Blanton had broadened his judgment of defective speech; or the latter survey was more painstaking.

An increasingly liberal opinion as to what constitutes defective speech is reflected in more recent reports. In two surveys by Dr. Stinchfield in 1925 (6) it was reported that of the entering students at Mt. Holyoke in 1922, 18% had defective speech and another 19% were required to take speech work as sophomores. In 1923 the corresponding percentages were 16% and 24%. In an intensive study of 1661 entering students in the State University of Iowa in the years 1930–31 and 1931–32 it was reported by Mr. Mabie (4) that "more than half (50.9%) of the students included in this inquiry are laboring with one or more specific handicaps of voice and speech." In his discussion Tuesday afternoon, Dr. Kopp pointed out that of students entering the College of Education at Wayne University 31% or 32% are held for remedial work in speech or voice.

A note of confusion is thrown into the picture by a recent report of a survey by Dina Reese Evans in the Cleveland Heights High School (3). Investigating the speech of 224 students in the 9A grade she reported three students with defective speech and none with defective phonation. However, she says, "Pronunciation and enunciation in 92, or 43%, of the cases was noticeably slovenly and inaccurate. . . . Six foreign accents were discovered." What is defective speech?

When we consider the general usefulness of the various surveys we see that the studies of the number of speech defects at the first year college level are useful to indicate the large number of children passing through twelve years of schooling and coming out with defective speech. They do not tell us, however, what to expect at the lower grade levels. The usefulness of the speech surveys below the college level is impaired by one or more of four reasons: 1, the method of investigation by reference of students by non-speech teachers is used; 2, a questionnaire method is used; 3, the cases are seen with such rapidity as to preclude thoroughness; 4, the basis of judgment of investigators apparently varies greatly. If a personal interview method is used and if the basis of judgment is constant we can in time arrive at some understanding of the relation to incidence of speech defects of grade level and environmental conditions.

In an attempt to answer some of the criticisms raised the author has undertaken two speech surveys one of which is completed, the other in process. During 1937–8 a survey was made of a random sampling of the 9A class in Central High School, Kansas City, Missouri (5). A personal interview was held with each of 178 students. The interview was of sufficient length to check both conversational

speech and oral reading. There was a recheck of all cases classed as defective, together with the Educational Counselor of the School. Cases not considered as defective by the Counselor were omitted from the defective group. Practically no changes were made due to the re-examination. To afford a basis of judgment the following suggestions were given to the Counselor before the re-examination.

By defective speech we mean speech sufficiently different from "normal" speech to call undesirable attention to itself in ordinary conversation, in conference, or in a more formal speaking situation. "Normal" is here used to describe speech ranging from the slovenly speech of the street to the precise diction of the stage or radio. By defective voice we mean a voice unpleasant or inadequate to a degree sufficient to give us a negative reaction toward the individual.

The high school student is receiving training that, directly or indirectly, is designed to enable him to function in a social environment on a more or less intellectual level. Many high school students are looking toward a life in business or one of the professions. When the student speaks, if his speech or voice is such as to handicap him in social situations, in business or professional activity it would seem reasonable to consider that speech or voice as defective. If he is basically unequipped to properly employ in a talking world the learning and techniques he is acquiring in school, he is to that extent fundamentally defective.

The speech defect may be evidenced in various ways. There may be omission of sounds, substitution of one sound for another, faulty production of a sound or sounds, or meaningless repetition of sounds, words or phrases. There may be interruptions in the rhythmic flow of the speech without relation to logical content, or substitution of words of different meaning than called for by the context, etc. The person may be defective in articulation, or may stutter, or may suffer from aphasia. Perhaps a nice final criterion is this: is the phenomenon in question an evidence of carelessness, a thing the speaker can change at will, or is it something beyond his immediate control? The vocal defect may be in the nature of nasality, breathiness, sustained high pitch, raucousness, insufficient loudness, excessive loudness, etc. The criterion may well be: can the individual eliminate the difficulty at will without individual guidance?

The final results of the survey show that of the random sample of 178 high school sophomores examined, 14% were found to have defects in voice and/or speech. Of these students 7.9% were classed as mildly defective, 5.6% as having defects of medium severity, and .5% as severely defective. There is no difference of statistical significance between the normal and speech defective groups in intelligence nor scholarship achievement. The difference for the two groups of correlation between intelligence scores and scholarship index is not statistically significant. There is no sex difference in the number of articulatory defects. More boys have defective voices and more boys stutter. Foreign nationality background, nor foreign language influence in the home or environment seem of importance etiologically. The problem is of especial significance locally since 84% of the defective students have spent their entire school life in Kansas City. About three-fifths of the group were unaware of the fact that their speech or voices were not perfectly normal. More than three-fifths of the group have received no systematic instruction in speech either in school or privately. Two-fifths of the group have failed to take part in any kind of speech activities in their school career.

The relatively low number of speech defects found in this group

can be attributed to three reasons: 1, the interview in each case was too rapid; 2, the basis of the judgment was too severe; the investigator was attempting to judge from the viewpoint of the casual observer; 3, there has been for years a speech improvement program in the Kansas City schools.

The second survey to be reported is now in progress, a survey of speech defects in the Laboratory School of Indiana State Teachers College, Terre Haute. The survey will include all students from the kindergarten through high school (and all students of the College). The purpose of the survey is to learn exactly how many students have defective speech, just what students are defective and in what way the student's speech is defective. It is to serve as a basis for a whole approach to a school problem. The method of the survey is a personal interview with each student.

This preliminary report is of the 208 students in grades 3A to 7A already examined. Of these 208 children, 45% were found to have defects in voice and/or speech. Of these students 39.5% were classed as mildly defective, .5% as severely defective. Of the 208 seen, 86 have articulatory defects, 13 have defects of voice and 5 have speech defects (stuttering and general oral inaccuracy). There is no appreciable difference in intelligence between the defective and normal. The average I. Q. of the normal is 101.1 and of the defectives 102.4. More boys than girls have articulatory defects. More boys than girls have defective voices. Other disturbances of speech are about the same in boys and girls. There is only one stutterer—a girl. Of the 86 persons with articulatory defects, 14 have a background of foreign language influence. The most common voice defects are breathy voice and hoarse-husky voice. The most common speech sounds that are defective are, in this order: s, z, ʃ, ʒ, tʃ, dʒ, h. Only one student of all examined was aware of any defect in his speech. Of the 94 students found to be defective in speech and/or voice only 8 were reported defective in response to questionnaires sent out prior to the personal interviews. It is important for us to realize that superior classroom teachers have not been trained to even recognize speech defects.

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FAMILIAL INCIDENCE IN STAMMERING

JOSEPH M. WEPMAN
The University of Chicago, University Clinics,
Speech Clinic

Introduction

The literature of speech correction is replete with statements by learned authorities both here and abroad attesting to their opinion that stammering is inherited (1). There is no record in the literature, however, of any definite attempt to prove this assertion. There have been studies made, notably those by Dr. Bryng Bryngelson at the University of Minnesota (2), coupling the inheritance of stammering with the inheritance of sidedness. Like other similar studies no controls were used for comparison and there was what appeared to be an attempt to prove an assertion already made. These studies have served to point a definite indication of the existence of an inheritable factor, however, and this study was made to prove or disprove as the case might be, the existence of familial incidence in stammering.

The Study

The incidence of stammering was examined in the backgrounds of two hundred and fifty (250) stammerers and a like group of non-stammerers*. The two groups were paired for age, sex, and social environment (within definite limits). The method used in selecting the families to be studied was to take the stammerers as they appeared in the clinics or as they were suggested to the author and pair them with completely unselected non-stammerers. The law of statistical regularity according to Jerome's Statistical Methods (3) reads, "that a moderately large number of items chosen at random from among a large number are almost sure, on the average, to have the characteristics of the larger group." This is the basis for most computations of social statistics being known as random sampling of the population.

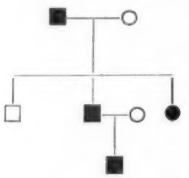
In collecting the tabulated information concerning the family histories of the selected individuals, the author personally interviewed the parents and solicited known facts concerning the ancestry. Wherever there was doubt, or one of the parental pair was without information, or wherever stammering was present in the immediate family and could be imitated, the record was not used. No attempt was made at finding predetermined information. The only facts elicited concerned the number of stammerers, their sex, and their position in the family tree. The results as here compiled, then, represent, what the author believes to be, reliable statistics concerning the incidence of stammering. If there be error, the assumption is,

that it is on the positive side rather than the negative, for the average person would deny rather than affirm the presence of stammering in the background, as a matter of family pride. Family charts were made for each family investigated showing the position, age, and sex of each stammerer and their immediate families. (Chart I.)

CHART I

TYPICAL FAMILY HISTORY SHOWING THREE STAMMERERS IN BACKGROUND

Stammerer's Name S. G. Age 10 Sex Male



Legend

○ Female

□ Male

Black represents incidence of stammering.

Cases for this study were obtained from the speech clinics at:

1. University of Chicago.
2. Central Free Dispensary in Chicago.
3. Michael Reese' Mandel Clinic in Chicago.
4. University of Wisconsin in Madison, Wisconsin.
5. The Public School System in Whiting, Indiana.
6. The author's private office in Hammond, Indiana.

The following three charts represent, numerically, the accumulated data. Each chart is composed of two parts, (a) the stammerers' backgrounds and (b) the non-stammerers', or the controls', backgrounds. Comparative ratios are shown with each chart, one ratio showing the number of males to females and the other ratio comparing the number of stammerers found in the backgrounds of the two groups.

CHART II

A. Stammerers	Cases Studied							Total	
1. Family type showing number of stammerers in each background...	6	5	4	3	2	1	0		Mean 1.40.
2. Number of families investigated.....	1	3	7	21	39	63	46	180	P. E. .077.
3. Total stammerers found (1 × 2).....	6	15	28	63	78	63	0	253	Ratio Male
4. Female stammerers...	2	3	5	12	18	11	0	51	to Female 4:1.
5. Male stammerers.....	4	12	23	21	60	52	0	202	

CHART II—[*Continued*]

B. Non-stammerers	Cases Studied					Total	
1. Family type showing number of stammerers in each background...	4	3	2	1	0		Mean .211.
2. Number of families investigated.....	1	3	3	19	154	180	P. E. .029.
3. Total stammerers found (1 × 2).....	4	9	6	19	0	38	Ratio Male to
4. Female stammerers...	1	2	0	3	0	6	Female 5.3:1.
5. Male stammerers.....	3	7	6	16	0	32	

Mean Difference, 1.16.
P. E. Difference, .082.
Ratio Mean A: Mean B as 6 : 6 : 1.

Chart III shows the same facts for the female stammerers' backgrounds and for their paired control non-stammerers.

CHART III

A. Stammerers	Cases Studied					Totals	
1. Family type showing number of stammerers in each background...	4	3	2	1	0		Mean .801.
2. Number of families investigated.....	2	3	7	26	32	70	P. E. M. .089.
3. Total stammerers found (1 × 2).....	8	9	14	26	0	57	Ratio column
4. Female stammerers...	1	2	4	5	0	12	5 : 4 as 3.7 : 1.
5. Male stammerers.....	7	7	10	21	0	45	

B. Non-stammerers	Cases Studied					Totals	
1. Family type showing number of stammerers in each background...	4	3	2	1	0		Mean .271.
2. Number of families investigated.....	0	1	4	8	57	70	P. E. M. .027.
3. Total stammerers found (1 × 2).....	0	3	8	8	0	19	Ratio column
4. Female stammerers...	0	0	2	2	0	4	5 : 4 as 3.75 : 4.
5. Male stammerers.....	0	3	6	6	0	15	

Mean Difference, .430.
P. E. Difference, .093.
Changes in 100 of Difference being a true difference—100.
Ratio Mean A: Mean B as 3.21 : 1.

Chart IV is a composite of the information shown in Charts II and III.

CHART IV

COMPOSITE

MALE AND FEMALE

A. Stammerers	Cases Studied							Total	
1. Family type showing number of stammerers in each background...	6	5	4	3	2	1	0		Mean 1.24.
2. Number of families investigated.....	1	3	9	24	46	89	78	250	P. E. M. 0.066.
3. Total stammerers found.....	6	15	36	72	92	89	0	310	Ratio
4. Female stammerers...	2	3	6	14	22	16	0	63	5 : 5 as 3.92 : 1.
5. Male stammerers.....	4	12	30	58	70	73	0	247	
B. Non-Stammerers	**Cases Studied**							**Total**	
1. Family type showing number of stammerers in each background...	6	5	4	3	2	1	0		Mean .228.
2. Number of families investigated.....	0	0	1	4	7	27	211	250	P. E. M. .012.
3. Total stammerers found (1 × 2).....	0	0	4	12	14	27	0	57	Ratio
4. Female stammerers...	0	0	1	2	2	5	0	10	5 : 4 as 4.7 : 1.
5. Male stammerers.....	0	0	3	10	12	22	0	47	

Mean Difference, 1.012.
P. E. Difference, .067.
Changes in 100 of difference being a true one—100.
Ratio Mean A: Mean B as 5.44 : 1.

Summary

Of the two hundred and fifty families of stammerers studied, one hundred and seventy-two (172) showed some incidence of stammering, while seventy-eight (78) did not. A ratio of 2.2 : 1 or 68.8 per cent of the families showed the trait. In the non-stammerers families thirty-nine (39) showed some incidence of stammering whereas two hundred and eleven (211) did not, a ratio here of .18 : 1 or 15.6 per cent showing the disorder.

Statistically the results obtained are reliable, since a difference to be reliable must be at least four times its probable error. In this study the various differences were from four times to twenty times as great. Since it is also true that the reliability of a difference

increases as the ratio of the difference to its probable error the possibility of pure chance creating the condition as noted is practically negligible.

The foregoing charts seem to express these pertinent facts: (1) that stammerers do tend to appear more frequently in the families of stammerers than in the families where there has never been stammering (in the ratio of 6.12 : 1) as has been predicated without statistical proof many times, (2) that stammering appears more frequently in the male of the species than in the female (ratio of 4 : 1). That stammering is more prevalent in the male is quite generally accepted and much has been written concerning it. Dr. Robert West (4) in his resumé of the White House Conference Report placed the ratios between the sexes at from 3 : 1 to 5 : 1, Dr. Travis (5) notes the ratios at from 2 : 1 to 10 : 1.

Conclusion

The data herein reported should lend weight to many considerations concerning stammering. For instance, that certain individuals in the general population are stammerers because of incidence of that disorder in their family backgrounds; that others are probably potential stammerers, but for some reason, at present unknown, do not stammer; that certain others, before this year is out, will become stammerers. It may be that stammering inherited duplex, from both sides of the parental line, is permanent and cannot be successfully treated, or that stammering inherited, simplex, from one parental branch, is amenable to treatment. This line of thought can be extended almost indefinitely, further research is necessary before it would prove of value. It is true, however, that if stammering be inherited, treatment can effect only the present manifestation of the disorder and cannot effect the cause. In any case, the extent of our present knowledge is too meagre to allow more than conjecture at the present time. It is hoped that this study will provoke further research along the lines here indicated, that since the facts here gathered definitely indicate a true trend, such studies be integrated and the results of large numbers of family histories be published. The author does not wish to take a dogmatic stand as to the inheritance of stammering but considers the facts as gathered to be of importance and of wide interest. Any family histories of stammering will be received with deep appreciation.

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LANGUAGE AND SPEECH HYGIENE, AN APPLICATION OF GENERAL SEMANTICS. By Wendell Johnson (State University of Iowa). 1330 E. 56th St., Chicago: Institute of General Semantics Monographs, No. 1, pp. 54, 1939. $1.50.

This monograph not only represents the conclusions of Professor Johnson after ten years' research on the problem of stuttering, but represents the most able expression of the point of view of Iowa since it has turned away from the cerebral dominance concept. Outlined are the principles of "General Semantics" giving particular attention to the prophylactic and therapeutic techniques. The fertile research mind of Professor Johnson, that has resulted in numerous publications, especially in this journal, is not only interested in alleviating stuttering and language disorders, but also in being certain that the psychology of language is a true science. With this in mind, he has developed his very important *Measures of Language Behavior* (Chap. 2). The core, around which "General Semantics" is concerned, Professor Johnson defines as the study of *word-fact* relations. The book represents the outline of Professor Johnson's course called, "Speech and Language Disorders" or "Speech Hygiene." This is undoubtedly one of the most important books of the year.

C. H. V.

SPEECH CORRECTION IN CEREBRAL PALSIES

(From the Standpoint of the Neuromuscular Specialist)

M. E. Pusitz, M. D.*
Topeka, Kansas

INTRODUCTION

The treatment of cases of cerebral palsy offers a most fertile field, and one which has been but little explored. There must be organization of material, however, so that incorrect and unnecessary data may be discarded, and all valuable contributions be made available. Many mistakes have been made in the past, but they have helped build up a proper concept, not only of the condition, but also of its treatment. For correct therapy, regardless of whether it is the work performed by the orthopedist, physiotherapist, gymnast, occupational therapist, speech correction expert, or psychologist, a proper understanding of the condition is essential, and of its manifold variations. There must also be the proper interplay of all the specialties, with the neuromuscular specialist as the pivot. In this, the speech expert plays a very important role, as long as he realizes his limitations.

DEFINITION

The term spastic paralysis has been used to cover this group of cases in the past. As Phelps and Zentay have stressed, this term is incorrect, since it applies to only one characteristic of the condition—spasticity, however, is the most frequent and constant symptom. Phelps uses the term, "cerebral palsy," which while inadequate, does cover the field. Zentay uses the term, "motor defective child." It is essentially an upper motor neurone lesion, affecting the cerebral motor centers. As a result, there is loss of cerebral inhibition—it is really not a paralysis at all.

INCIDENCE

There are no accurate statistics, but surveys which have been made, in some instances, show figures which exceed those of infantile paralysis. In Kansas, a state which investigates very inefficiently cerebral palsies, of 4617 orthopedic cases, 9.46 per cent were due to spastic paralysis. These figures are low, since the stage is well set for the investigation of all cases of infantile paralysis, but not so in

*Orthopedic Surgeon, Kansas Crippled Children Commission, Capper Foundation for Crippled Children.

spastic paralysis. In the author's practice, more cases of spastic paralysis than infantile paralysis are seen. The reason for these figures being so low are as follows. The social worker feels that these cases are for the neuromuscular surgeon, or the mental institution, labelling them as idiots or morons. Crippled Children Commissions are not anxious to admit these cases, due to the lack of uniformity in treatment amongst the various Orthopedic Surgeons. The parents are at a loss to know what to do, because the family doctor tells them that nothing can be done, while the quack tells them that he can cure the condition. The result of this is failure from all angles. Many of the milder cases where the child seems unable to hold up his head at the proper age, where he seems slow in walking or developing a sense of equilibrium are labelled as due to rickets, scurvy, or glandular deficiencies, when actually they are a type of cerebral palsy. There are also many cases of encephalitis and hemiplegia in adults, not included in these statistics. If all of these cases are included, then one has a much larger field for rehabilitation than in infantile paralysis. When it is realized that these cases require more specialized and prolonged treatment than the latter, taking many years to effect, that fully seventy-five per cent of these cases have some degree of a speech disturbance, and that marked improvement follows proper therapy in fifty to seventy-five per cent; it can be realized how important a field this is for the speech correction expert.

ETIOLOGY

The condition may be considered as congenital or acquired, the majority being the latter. The most common cause of the congenital type is due to lack of development of the pyramidal tract, leading to atrophy or sclerosis of the lateral columns of the spinal cord; to large cerebral defects; to porencephaly; to hemorrhage; or intra-uterine softening; also congenital syphillis is the cause of a small minority of cases. Intra-uterine infections, causing fetal encephalitis may produce more or less extensive destruction of the central nervous system, resulting in a similar condition, but these cases cannot be distinguished later in life from those where the injury occurred at birth.

The acquired cases are those occurring during labor, or those coming on later in childhood. Jones and Lovett consider intracranial hemorrhage, due to trauma of the child's head during prolonged labor, forceps delivery, or prolonged asphyxia at birth as causes. The hemorrhage is usually meningeal, but at times intracerebral, and gives rise to chronic meningeal encephalitis; sclerosis; cysts; atrophies; and porencephaly. Of those cases occurring after birth, one can list hemorrhage, embolism, endoarterial and periarterial changes, encephalitis, cerebral venous thrombosis, and chronic meningitis as the productive factor. There is a variant of poliomyelitis, which may involve the cerebrum, resulting in an admixture of spastic paralysis with flaccid paralysis.

PATHOLOGY

CLASSIFICATION. Zentay has presented a very useful classification of the central motor defects as follows:

"1. Pyramidal lesions (Spastic Paralysis); Monoplegia, Hemiplegia, Diplegia, Tetraplegia.
2. Extrapyramidal lesions (Hypermotility); Hemiathetosis, Bilateral athetosis.
3. Cerebellar lesions (Ataxia disorders); Hemiplegia, Diplegia, Tetraplegia."

Regardless of the etiology, the resultant clinical and neurological syndromes will depend upon which one of these three motor systems is particularly involved.

Developmental Anomalies. There is not only a lack of development of cortical regions, but even of parts of the brain. These cases have no intelligence and are not to be treated. There are other types where damage to the brain may be very extensive, resulting in a small percentage of the group being composed of idiots and morons, but it is impossible during the first year or two of life to distinguish these cases from the more intelligent ones. Certain acts of the newborn are really automatic and require no intelligence whatsoever. Investigations have shown that the normal activities of the newborn infant are reflexes controlled by the spinal cord, hence the difficulties of diagnosis are apparent.

PYRAMIDAL LESIONS. True Spasticity. The lesion involves the cortex or the subcortical areas of the parietal lobe of the heterolateral hemisphere. This is by far the commonest lesion encountered, and yet the number of cases which present spasticity alone is small, because this is usually combined with some athetoid involvement, which may be due to concomitant lesions in the frontal lobe or an extrapyramidal lesion. Delay in the development of motor functions is seen and sitting up may develop one or two years later than in the normal. There is hypertonicity of the muscles, varying from slight irritability to spastic rigidity. The limb is rendered useless, not because of paralysis, but from increased muscle contraction, and lack of co-ordination.

EXTRAPYRAMIDAL LESIONS. Athetosis. Although it is believed that certain lesions of the frontal lobe may cause athetoid movements, the basic pathology is found to be involvement of the basal nuclei, particularly the caudate nucleus (striate body) on one or both sides. In this type of lesion there is resultant athetosis, tremor, inco-ordination, etc., and muscle tonus may be actually decreased.

CEREBELLAR LESIONS. Ataxia. Here the lesion is in the posterior fossa above the tentorium. There is poor equilibrium, co-ordination, and tonus regulation. Muscle tone may be decreased, and ataxia in these cases may be due to both sensory and motor involvement.

NEUROLOGICAL DIFFERENTIATION

REFLEXES. In the *Pyramidal lesion*, or true spasticity there is hyperactive knee and ankle jerks, a positive Babinski sign, occasional ankle clonus, diminished or absent cremasteric and abdominal reflexes, and hyperactive biceps, triceps and periosteoradial reflexes.

In *Extrapyramidal lesions*, or athetosis, the reflexes may be normal. A Babinski sign may be present. The cremasteric and abdominal reflexes and arm reflexes are normal. (It is easy to mistake a reflex that is being reinforced automatically by involuntary motion for a true hyperactive reflex; one should therefore exercise great care in the examination of reflexes.)

In *Cerebellar lesions*, or ataxia the reflexes are not particularly affected.

MUSCLE ANALYSIS. Muscle spasm and sensory impairment cause gross clumsiness, while inco-ordination due to cerebellar involvement, and athetoid movements complicate the picture. Increased tone may be localized to one or a few groups of muscles, but it is usually general with no uniformity of involvement. Careful muscle examination will reveal some muscles which are extremely spastic, others which seem almost normal and still others which may be flaccid. Similar charts may be made of these muscles as those for infantile paralysis, thereby giving an accurate picture of the balance of muscular activity.

SENSORY DISTURBANCES. There is more or less a loss of appreciation of the relative intensity of stimuli. Fine differences of heat, cold, and touch, are imperceptible. Appreciation of spacial relationships may be lost, thus he may be unable to identify the precise parts stimulated, (tactile localization). Discrimination between two points of stimulation (tactile discrimination), may be affected. Muscle spasm itself upsets the normal proprioceptive impulses from both muscles and joints, and this must always be considered, since inco-ordination is of sensory origin to some extent, especially in the pure spastic type.

NEUROLOGICAL STATUS. There is no paralysis; messages are still relayed to the muscles, but there is no longer the controlling, co-ordinating action of the higher centers. There is no repetition of an impulse traveling along one set pathway to its termination. Instead, when the message is sent out from the brain, the impulse breaks out with an explosive effect, following no set nerve pathway, but seemingly breaking up into subdivisions which suddenly fly forth, criss-crossing, and reaching many terminations at the one time. Instead of localized muscle action, there is generalized action, in which agonistic and antagonistic muscles may contract at the same time For example, in the attempt to flex the wrist, instead of this, there is contraction of both flexors and extensors. Not only so, but the muscles of the fingers, the hand, the arm, and even of the trunk and the head; in fact, the whole patient may be thrown into a violent paroxysm of muscular contractions, as a result of the simple message from the brain to flex the wrist.

Another defective muscular mechanism which occurs, follows the general laws of muscle action. If a muscle is set to perform a movement of which it is incapable, it will not even attempt to do so, and that is not all, it will rely upon its synergist to perform the action required. The result is that the opposite movement is created. Movements are represented on the cerebral cortex, and not individual muscle action, therefore a cortical impulse leads to contraction of muscle groups, and not of individual muscles. With overactivity of the adductor muscles, if abduction is attempted, the deltoid will make no contraction; but instead the adductors will contract to assist in elevation of the arm by means of scapular movement. In the same way, if the patient attempts to make a fist, as a result of a volitional impulse, the arm may be firmly adducted; the forearm flexed, and held rigid; the wrist dorsiflexed, or even volarflexed; and the interphalangeal joints fully extended by the interossei muscles—nothing whatever may happen in the direction of the flexion of the fingers. If this is allowed to go on, then the physical barrier is insurmountable. The above considerations are very important in treatment. It is essential to understand component muscle action. If the flexors of the wrist are very powerful, every attempt to make a fist, may result in flexion of the wrist as the first step of the movement. This creates a muscle insufficiency in the flexor muscles of the fingers, the latter going into hyperextension. The key to the understanding of this problem is in the recognition of the preponderance of wrist flexor action.

INTELLIGENCE

The common impression is that cerebral palsy cases are deficient in intelligence. The older text-books, for example Gower's, considered them all as mentally deficient prior to 1900. In 1923 Jones and Lovett agreed that some degree of mental deficiency is present, although in a number of cases this was hardly noticeable. They noted that when these cases came to adult life they usually showed some impairment of mental stability (not mental deficiency). In 1935 Stevenson defines the condition as, "the one well known disease of the pyramidal tracts with little or no mental defect." In 1938, Mills classified five out of two hundred severe cases as idiots. There is no doubt that the more apt students of this condition believe that by far the vast majority are mentally intelligent. If one adopts a proper method of estimating this intelligence, the author believes that 50 per cent are of average intelligence; 5 to 10 per cent are abnormally bright; about 10 per cent or less are idiots or low morons; and the rest are substandard or high moron individuals.

The spastic has a peculiar, stupid look, being unable to register emotions, due to the inco-ordination of the muscles of expression. The mouth may be wide open with drooling of saliva, and they cannot learn to express themselves in speech like others can and do. Unless their environment is exceptional, their visual and auditory vocabulary must be small. When one adds to this already, almost unhuman

looking person choreic or athetoid movements, it can readily be seen why so many look upon them as mentally deficient. The usual mental or intelligence tests, such as the Binet-Simon, are inadequate for these individuals. There must be some tangible method of approach, which does not depend upon vocabulary, but upon certain acts. The most important criterion of whether a case is, or is not intelligent is whether progress can be made with proper therapy.

SPASTIC PERSONALITY. It is frequently stated that there is no such thing as a "Spastic Personality." The author disagrees with this assertion, and places the spastic individual in a class by himself. There is not only hypertonicity of muscles, but a whole complex of physical abnormalities, due to lack of cerebral control, complicated by athetoid movements and inco-ordination of the entire body. The author summarizes in his book as follows:

> Owing to the physiopathological set up, there is not the same opportunity for the development of "nerve pathways" (the author does not desire to enter into the pro and con of "nerve pathways" with the psychologist—this is merely used for illustration). The activity of the muscles do not result in a harmonious score, but in discord. The result is chaos for the spastic as soon as he develops enough intelligence to consider himself and his surroundings. The age group at which this occurs seems to be four to five years in the more intelligent cases, possibly some years later in the less intelligent. This conclusion has been arrived at through the questioning of some forty older spastics in the adolescent or adult stage. The earliest impression on the mind of the spastic must be frustration of effort or desire. Then fear and worry develop as soon as the intelligence required is reached. As a result of the tremendous amount of effort, both physical and mental, required for the production of any movement, the first tendency in these cases is to not try to do anything, since when certain movements do take place as a result of mental thought, or through stimulation, there is bound to be discord. It soon becomes evident to the spastic child that he is not considered in the same light as other children. In many instances, parents talking in front of the child will make all sorts of pessimistic remarks; will be oversolicitous, and all this can have but one effect, namely, to make the spastic realize that he is not part and parcel of the run of things. He may, as a result, develop the tendency of all cripples, take to the background; or he may in fact, be forced in the background through the unintelligent handling by other individuals. Picture the feelings of the severe spastic when he sees some handsome young boy or some beautiful young girl recoil in horror when they gaze upon him. This spastic may be hungry for the association of these other children, purely from the instinct of getting together with people, or he may, unknown to himself, be expressing a sexual desire which cannot be satisfied. Be that as it may, it is not long before the spastic realizes that theoretically only is he free—practically he has been ostracized from society.

THE GENERAL TREATMENT

DIFFERENTIAL DIAGNOSIS. It is absolutely essential that those who treat these cases be fully cognizant of their differentiation and analysis. It is not simply a question of spasticity, but rather an analysis of all the motor systems. This is not only important in the general treatment, but particularly so in speech correction. It is essential to distinguish between hypertonia of muscles and hypotonia. In the former, bracing is contraindicated, whereas in the latter it is very much indicated. Developmental cases give a very poor prognosis, and usually comprise the unintelligent group. In the cortical

or true spastic type, the prognosis is much better, and repetitive methods are of great value in this type. In athetoid cases, however, the prognosis is not so good, and repetitive methods are of no great value. It is very essential to induce relaxation before any worth while progress can be made. In cerebellar cases, and in fact all types it is very essential that the sensory education precede the motor. It is difficult to differentiate between spasticity and athetosis, and yet it is very important to do so. Spasticity is due to a hypertonicity or stiffness of the muscles, as a result of which there is lack of co-ordinated motion, while athetosis is an involuntary form of motion. Thus in the spastic type, the attempt to produce a certain movement creates the stretch reflex, causing, secondarily, contraction of other muscles, resulting in inco-ordination. In athetosis, on the other hand, there is involuntary movement to begin with, and when attempts are made to control this involuntary motion, other muscles go into contraction, resulting in inco-ordination. It takes much experience to differentiate these types.

In true spasticity, therefore, the defect is rather uniform, and the type of speech utilized by truly spastic subjects can be learned easily by those who work with them to any extent. It is due, of course, to definite spasticity in some part of the speech mechanism, and the result is speech which is defective, but in definite ways. In the athetoid patient, where involuntary motion is the primary difficulty, speech is upset by the involuntary athetoid movements of; for example, the tongue, and since these involuntary motions never coincide with the voluntary ones made by the patient when he speaks, the words never come out distorted in the same way. On repeating the same word over and over again, it is given out differently each time. No amount of improvement can ever occur in the severe, true, athetoid case without some relaxation induced as a prerequisite. Cerebellar speech is an ataxic speech. It is an explosive, hesitant type of speech, scanning in character, with stretching of syllables which are sharply cut off from one another. In this type of speech it is very important to correlate proprioceptive impulses with definite motor movements.

It can therefore be seen that study of the pathological and clinical features of this group of cases demonstrates the usual methods of speech correction work as entirely inadequate. It is true that the presence of the normal gag reflex, chewing reflex, swallowing reflex, etc., is inductive to normal speech. But in these cases of cerebral motor defects, it is extremely important that there be a conditioning of inhibition of increased muscle spasm, the induction of relaxation with lessening of athetosis, and the prevention of multiterminating impulses. The more voluntary effort the patient puts forth, the greater the emotional disturbance, the more is the athetosis exaggerated. Not only is there mental exertion, but there is also physical exertion causing fatigue. The tremendous activity of the muscles demand an increased metabolism. The spastic uses up more energy in one attempted motion than the normal does in one hundred such

motions. All of these factors have to be taken into consideration in the approach.

It goes without saying that before speech training proper is commenced, the sensory balance should be investigated. Auditory disturbances and visual disturbances should be ruled out. If these are present, then every attempt should be made to investigate and treat them. Since these principles are so well known to the speech expert they need not be considered further.

General Factors. While the author agrees with Carlson, that in the early stages, it is best to segregate the spastic from normal children, this is not feasible with the set up as it is at present. The next best thing is the formation of clinics, primarily interested in the treatment of cerebral palsy cases. At the head of such a group there must be the neuromuscular specialist, aided by a competent physiotherapist, occupational therapist, and speech correction expert.

It is very essential that treatment be instituted as soon as the diagnosis is made. It is possible even in the very early years to begin definite training. It should be remembered that there is a tendency for the normal to develop walking by the age of two, and speech by the age of five. Granting that the spastic is delayed a year or two, or three, one should try as much as possible to develop the same acts in equivalent age groups. To allow these cases to go untreated only encourages the formation of deformity, contractures, and physical and mental deadlocks. If no special school is available it is best that the child be kept at home. To enter these children into schools with normals is but to invite mental deadlock. They cannot keep up with members of the class for one thing, and they are open to the ridicule of others.

The general health of the patient is important. There should be adequate rest. It is essential that proper and sufficient food and nourishment be available. The intake of Vitamin D and calcium is theoretically important in lessening irritability. Ultraviolet light is also useful.

The Psychological-Psychiatric Approach. Obviously, the mentally deficient cannot be treated. Mental deadlock starts almost at the beginning of the mental existence of the patient.

Considering those cases taken for treatment every effort should be made to have the patient feel himself part and parcel of the run of things. He should not be neglected, but should be given the same consideration as normals. In fact, he should be made to feel that as much is expected of him as others. However, treatment must not be pushed beyond his capabilities. More time is required for their nervous system to make differentiations, resulting in a limit to their capability, depending upon anatomical and physiological factors. Physical barriers are also capable of producing mental deadlock, and this has been shown by psychological investigations. For example, to tie the left hand behind the child's back, in order to make him use the right hand, is but to invite mental deadlock, as a result of a physical barrier, with development of speech defects, mental behavior

patterns, etc. On the other hand, mental deadlock is capable of increasing physical distress; hence there must be a proper combination of physical and mental measures.

Carlson has made an excellent contribution in his principle for reduction of stimuli. He states:

> Those natural or environmental stimuli which affect the normal individual, also affect the cerebral palsy. Indeed it is indisputable that the cerebral cortex receives these stimuli; visual, auditory, proprioceptive sense, etc., summates the stimuli, and transfers them to their proper channels for their final release through motor or other pathways. In the cerebral palsies where there is considerable damage to the cerebral cortex and the ideational centers are preserved, these stimuli affect the injured brain in a manner shown in the individual cases. As shunting of one or more sensory stimuli will affect the cortex in degrees, the final release will be somewhat more towards the normal, as for example, the spastic athetoid cases who have great difficulty in doing simple acts like feeding themselves, and yet are able to do skilled acts satisfactorily, like playing golf, playing the piano, and working out laboratory problems in chemistry and bacteriology. With educational training, new channels of release will be provided, a partial inhibition of sensory stimuli will be provided, a partial inhibition of sensory stimuli will be accomplished, and in due time a summation of the sensory stimuli will result in more or less the normal manners.

This concept of Carlson's is very useful as an adjunct. It should not, however, occupy the whole span of attention in treatment. It is a good policy to have the spastic focus his vision on a certain point in his environment, so as to cut out all other unnecessary visual stimuli. Thus in walking, he should look straight ahead of him, and not at his feet; in shaking hands, his vision should be focused on the hand which he desires to shake; and in speech, his vision should be focused on the lips of the person to whom he is speaking. Lip reading is a very valuable adjunct to all these cases.

Another important detail is to give the spastic individual a feeling of personal worth. Without this, there is no true relaxation in the individual as a whole, which is so important for inducing relaxation of the part.

PHYSICAL TREATMENT. In the treatment of a spastic case, the psychological approach is indeed very important but the physical side of treatment is just as important. The mistake that has been frequently made in the past is to consider this or that procedure, whether it be massage, manipulation or surgery, as the cure for spastic paralysis—in truth there can be no cure. The whole system of treatment is of a palliative nature only, and intended to mask the defects present, so as to enable co-ordinated muscle function to take place. In this regard, one must take into consideration the very basic pathology of the condition. One is not dealing with a case of pure spasticity alone, pure athetosis alone, or pure cerebellar involvement. One or the other of these motor systems may be particularly involved, but there is an admixture, the clinical syndromes depending upon which is the most prominent.

Regardless of which motor system is involved, as a result of the increased muscle spasm, and the sensory impairment, there is gross

clumsiness and inco-ordination. Involuntary movements of an athetoid nature complicate the picture still further. In order to develop co-ordinated muscle function it is essential: firstly, that the joint can be moved the required arc; secondly, that the proprioceptive sensations be directly correlated with movement. The first prerequisite in treatment is to remove all serious barriers to such movement. The nucleus in treatment is of a conservative nature. By means of heat, of proper degree, one encourages, if not actually produces relaxation of the part.

In order to produce relaxation of a part, it is essential that the patient as a whole be relaxed, which is the function of the psychological approach. The heat application is then followed by an effleurage massage, which has both a reflex and a mechanical effect. The mechanical effect is intended to rid the part of accumulated waste products. The reflex effect, however, is the more important and is sedative in character. It must be remembered that there are certain areas, especially the distal portions of the extremities, which are very sensitive. If the effleurage irritates rather than relaxes, then it should be altered. No heavy type of massage is indicated. Following the massage, passive movement is instituted, which should be gradual and sustained in character, working proximodistally, the more distal area being treated as the proximal is enabled to relax.

Proper therapy will soon produce a state of inhibition of the increased muscle spasm, which remains for a longer and longer period, even after treatment has ceased. This after period can finally become permanent. When this is so it can be said to be conditioned, and the muscles may be stretched without producing the stretch reflex.

The next step in the routine is the introduction of assisted movements, which at first are pendulum-like in nature. Gradually the amount of assistance is lessened. In these active movements, only the simplest should be introduced at first, then resistance and speed are added, making the procedure more and more complex. During this whole preliminary period of training, with each movement the attendant uses a command. It may be necessary that the child see another patient or the attendant perform the movement first. In this way the sensory education takes place so that the visual, auditory and proprioceptive sensations are linked together with definite motor acts.

The inhibition, when conditioned, readily spreads so that it involves not only the part being treated, but contiguous regions, and even the child as a whole. Thus with the conditioning of inhibition, it is found that the drooling of saliva and the incontinence of urine and feces are abolished. If the head, neck and face muscles are inhibited, and drooling ceased, speech returns to some degree without any training at all. It must not be thought that one simply goes over and over again these same movements aimlessly—there is an objective, so that the movements are directed along certain acts, such as

washing, dressing, or eating. In this way there is no waste of time in doing unnecessary gymnastics.

SURGICAL ROUTINE. It must again be stressed that there is no cure for spastic paralysis. Operations have been devised for particular objectives, and the most important is for correction of deformity or contracture. While it is true that with the conservative measures already considered one can, with persistance, correct deformity, this may be so difficult or take so much time and effort that it would be much more practical and economical to correct the deformity once and for all by surgery. The time can then be spent better by utilizing it for actual muscle re-education. Certain operations are directed against the exaggerated tonic spasms of the muscles, for example, Foerster's resection of the posterior roots. This particular operation would have no direct effect on athetosis, while Putnam's section of the extrapyramidal tracts of the spinal cord is specifically intended for athetosis, and it has no other value. It is also to be noted that as operations are carried out to relieve increased muscle spasm, there does seem to be a beneficial effect as well in lessening the athetosis indirectly. Finally there are certain operations intended primarily to balance muscle power about joints. The balancing of muscle power is only instituted if and when the patient has already had preliminary training, not only in the conditioning of the inhibition of increased muscle spasm, but also in muscle re-education.

Burman has recently introduced a method of treatment in which he injects curare, the Indian arrow poison, which paralyzes nerve endings and muscles. By injecting the drug in very small doses, it seems to have an affinity for the hypertonic muscles. This offers a very hopeful adjunct in treatment, but at best it can only be an adjunct and never the treatment itself.

Whatever measures are used, it should be realized that no one procedure is "the cure." The manifold problems presented must be treated one by one, although they are interrelated. The basic foundation in the routine of treatment is the conservative muscle re-education plan. All other measures are adjuncts, but the adjunct may be a most important item. With a flexion-adduction-internal rotation of the hip, conservative measures cannot be successful. In fact, with severe deformity, there is no possibility to move the joint passively, let alone actively. There is no opportunity to develop the sensory education. However, with the release of contracture by surgery, the physiotherapist can then start conditioning of inhibition of increased muscle spasm, and the development of proprioceptive education by passive movements. The muscle re-education still is the important nucleus—but it could not be started were it not for the surgical measure. The surgical procedure, however, is not "the cure" by any means.

Muscle re-education leads directly to the development of certain useful movements, also enhances and makes more permanent the conditioned reflex which has been instituted. In other words, one

does not have the spastic learn to play the piano by passing on from the simple movements of the fingers to the more and more complex, because this seems much more difficult for him than allowing him to go directly to the piano, attempt the complex movements from the start. But do not do this before he has had preliminary training in conditioning the inhibition. The principles of muscle re-education are very definite, but cannot be gone into in this treatise.

SPEECH CORRECTION WORK

The foregoing descriptions have demonstrated the interrelationship between physical barriers and mental disturbance. Physical deadlock can produce mental deadlock, with the production of mental behavior patterns, and speech defects, aside from the actual involvement of the muscles having to do with speech. If one adds to this, the effects due to athetosis and cerebellar involvement, one can readily realize that the problem is not as simple as in any other branch of speech correction work. It is true that if there is a normal gag reflex, swallowing reflex, and chewing reflex, speech correction work is not difficult. There are so many factors, however, in the cerebral palsy case, which act to upset these reflex mechanisms. It is senseless to expect to develop any degree of worth while speech in a spastic, unless there has been a preliminary period of training along the lines already suggested. The speech correction expert is making exactly the same mistake today that the orthopedist made years ago. One can just as well expect the spastic to get up and walk as to talk without this preliminary training.

The first step in speech training work has nothing at all to do with scientific speech work. Rather it has to do with the release of physical barriers, and the correction of deformities of the body as a whole, and the institution of those measures which allow the induction of relaxation, which will enable passive movements to be performed. The method correlates visual, auditory, and proprioceptive impulses with definite motor acts, through the inhibition of increased muscle spasm. This conditioned inhibition has a tendency to spread. Frequently the author has had speech correction experts ask whether there is any "carry over" effect in cortical activity. Clinically this can be answered in the affirmative, and this has been demonstrated in the discussion of the neurological status of the patient. It must be remembered that only acts, not individual muscle actions are represented in the cortex. Muscle groups, and not individual muscles are called into activity through volitional impulse.

The work begins in speech correction with the patient as a whole, but soon concentrates with the head and neck in particular. It is repeated that to simply attempt improvement of the reflexes, and then go on to the attempt at sounds is similar to trying to get the patient walking without preliminary training (through surgical intervention, or gymnastics, for example). It is true that in certain cases this is possible, but in the vast majority failure is the result,

with the development of further complexes, mental behavior patterns, and possibly even aggravation of the speech defects. On the other hand, the conditioning of inhibition has a tendency to spread. The removal of physical barriers in itself, improves the mental status of the patient, as well as his speech. With this conservative type of therapy, without any use of speech correction work, one finds that the patient who previously had inability to make intelligent sounds, not only becomes able to make these sounds, but to even speak some words. This does not mean that it will go on to normal speech, but it does demonstrate the value of this method of approach.

The first step in the program is therefore the approach of the patient along physical as well as psychological lines. Conditioning should first involve the patient as a whole, and then the muscles of the head and neck in particular. The speech correction expert should therefore fall into line, with proper timing tendency. The natural tendency is for the normal child to acquire the ability of speech by the age of five. Every attempt should be made so that the spastic child shall acquire this ability at an equivalent age, the spastic being retarded a year or two due to his condition. In true spastic cases this should be started at least by the age of two or three, after there has been conditioning, not only of the body as a whole, but of the head and neck. In cases with severe athetoid involvement, or cerebellar involvement the process will be much longer delayed. It is very essential that progress be made slowly and within the capabilities of the patient, so as to prevent development of a mental deadlock.

Not only should there be conditioning of the muscles of the head and neck, but those of the face, mouth, and tongue must be considered. Relaxation is necessary to allow passive movements, and elongation movements, tapping and petrissage, may be useful. From this one passes on to assisted movements, the assistance gradually being withdrawn, and then even resistance is gradually applied. The association of the visual, auditory, and proprioceptive sensations with active movements is to introduce function. The muscles of the mouth and tongue are subjected to the same routine of conditioning the inhibition of increased muscle spasm. It is not worth while trying to skip this phase of the work. These measures and gymnastics serve merely as an introductory measure for co-ordinated muscle function, and while not an actual step in speech correction work per se, they pave the way for the latter. The training of the tongue muscles should follow conditioning of inhibition in the head and neck. With proper routine, there is decrease or cessation of drooling or choking on swallowing. The patient soon learns to be able to use the muscles of speech in a more co-ordinated fashion.

When relaxation is possible to some degree, actual gymnastics of the lips and tongue can be instituted. These should not be persisted in, since they are not directly connected with speech, but they should be developed since they are indirectly connected. The movements at

first are assisted. Thus if the patient has the tongue protruded, but cannot flatten it, the masseuse uses the hand to flatten the tongue, or the patient may use the lips to flatten the tongue. The tongue is then flattened or pointed, swung from one region to another, up and down, and in and out. It is taught to touch the roof of the mouth, the various teeth, etc. All of these movements can be related to certain nursery rhymes. Complete control of these movements of the tongue is very difficult to obtain in severe spastic cases, and therefore these movements should not be persisted in to such an extent as to produce mental deadlock, since the goal may be so high that the patient may never be able to reach it. As a preliminary procedure to actual speech training, it is most useful.

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BIBLIOGRAPHY RELATING TO READING ON THE HIGH SCHOOL AND COLLEGE LEVEL. By R. Strang. New York: Teachers College, Columbia University. pp. 194, 1938. $1.50.

This almost two-hundred page partially annotated bibliography on reading should be on the shelf of every speech corrector. The material is divided into 19 chapters and each chapter is separated into whether the references are general, or refer to the high school or the college level. The bibliography is not comprehensive, but it does give an appreciation of the scope of the field, the names of investigators most active, the magazines in which articles are most likely to appear, a supply of ready references on each phase of the subject, and show the gaps where further research is needed. There are forty-three pages of references in the five sections on deficiencies.

C. H. V.

A PUBLIC SCHOOL APPROACH TO TREATMENT OF STUTTERING

CLARA B. STODDARD
Supervisor of Speech Correction Classes,
Detroit, Mich.

Every possible physical correction should be recommended before the child enters a class in speech correction. In the physical, one commonly finds balance disturbed; a poor poise; the body, the extremities, the eyes, all partaking of this general disturbance. These children should be placed under the special care of the physical director, who has carefully planned a series of corrective physical exercises to meet the needs of just such children. Folk dancing, esthetic dancing, and gymnastics, pure and simple, will train these weakened, underfed muscles to better response. The diet is arranged with a view to strengthening the weakened tissues.

In the mental field, one often notes a self-centered, rather egotistic individual who believes (whether consciously or not) that the whole world has its eyes fastened upon him. His reactions are abnormal. Morbid fears possess him and these emotions block the speech. Stutterers rarely look one squarely in the eye when talking. They complain that it hurts to look straight before them—that the constant shifting so common among them relieves this pain. Perhaps the trouble is physical, perhaps mental. We then place the child in the care of a psychiatrist, who probes the mind and gives wise and judicious counsel to eradicate the difficulties. Mental, as well as physical hygiene, has its place in our school system.

Children who have speech defects do not hear as well as perfectly normal children and the sight is often impaired. The finer, more delicate sound perceptions are often entirely beyond them. Sensory intake is poor and the motor output is below normal. We must find out whether there is a physical or a mental difficulty and act accordingly. The endocrine system is carefully studied, and physical and mental remedies given to the stutterer to aid his emotional control.

The body, particularly the extremities, may be poorly controlled. The tension of the muscles, the jerky inco-ordinate movements, are common to all afflicted with a lack of emotional control. Relaxation will relieve this strain. Games of all sorts change the tensions, relax tired muscles, and calm, quiet breathing relieves the strain in any part of the body. A calm, quiet body may help to produce a quiet mind.

The tones of voices of stutterers are weak, faulty and poorly modulated. Definite exercises should then be used to develop better control of the vocal mechanism. The child often loses his fear of hearing his own voice, when it becomes possessed of better quality and greater continuity.

Continuity is nearly always lacking. There is a very definite broken rhythm in the voice of the child who has a speech defect. The

trouble may be a poor auditory image of the vowel sound. Memory is poor and he does not quickly recall the image of the vowel so that the break may be definitely after the initial consonant. Then there may be a rapid enunciation of words or phrases, in which no decided break occurs—then the break again. So we must plan a very definite series of exercises to develop continuity of voice, memory of vowel formation, inflection, and rhythm. Memorizing good poetry and prose is invaluable here.

Speech response is lacking. These children are not awake, alert to the need of good speech. They are self-centered, perfectly satisfied with the halting, stumbling production. Sometimes they fail to recognize their lack of good speech. Their thoughts are not upon the subject in hand, but are usually far away in a dream land of their own making. They often fail to prepare their lessons, thinking they will not be called upon because of the speech difficulty. Then, if they are called upon, emotions block the speech simply because they lack knowledge of the subject in hand. Require them to recite in class and at least attempt to concentrate upon the subject matter. Show them that preparation is half the battle. They will speak better if they "know that they know." Give them a training in responsive exercises, those which shall require an immediate and exact response.

Music is of great value. It soothes, quiets, and is usually a thing they can do without great fear. If the same quality, direction of breath, volume, and precision of diction, which they use so naturally in singing, could be put into the speaking voices, the speech output would be much improved. There seems to be no over-innervation during the singing. Why not then use music as a bridge to better speech, trying to use as well modulated, well rounded speech notes as manifest in the singing tone? That should give children a very definite sensation as to the transition from the singing to the speaking voice, and a standard for tone, rhythm and continuity.

Stutterers may have two distinct and separate images of a single sound, one correct and one incorrect. This latter image is called up more quickly during emotional strain than the correct form. For that reason, a training in phonetics is valuable to strengthen the correct image. The vowel sounds do not appeal so much to the muscular sense as do the consonants. They must be associated first with the auditory images, and these must be persisted in until the child recognizes and reproduces the correct vowel sounds with their various shadings without hesitancy. He may be slightly tone deaf, and training may develop his ear mindedness. In the Detroit schools we have a very definite system for vowel and consonant drill, associating it with objects, pictures, words, and thoughts.

Stutterers' voices rarely have good volume. They are afraid of the sound of their own voices. They should be trained for speech in a room sufficiently large so that returning to an ordinary school room will not scare them. Many are apt to think speech training can be done in any little room that no one else uses, but this is a great fallacy and superintendents, supervisors and principals should see that the speech teacher has a place where a good, round, full delivery can be produced.

Oral gymnastics play a large part in the correction of oral inactivity. Judicious exercises for the development of the lips, gums, tongue, soft palate, pharynx, when given as play or for relaxation, train these organs so that they can respond when the demand is made upon them. Children enjoy these as much as a good play and are as much interested in making them respond to stimuli as they are to making the limbs respond to physical gymnastics.

Every teacher of speech correction realizes that the stutterer's control of respiration is very poor. Where oral inactivity prevails, the direction and quantity of breath is of great importance. As "breath is the material of which speech is made" (Bell) training must be given and continued until the child has self-control enough to not allow every passing emotion to interfere with the breath control. Without this training, the emotional disturbance may interrupt or completely block speech. Therefore, let every teacher deliberately train the breathing apparatus, so that it, too, can meet adequately the demands upon it. The breathing is the first thing affected by an upsetting emotion. If we keep allowing it to be interrupted, we soon notice a depression. Teaching easy breathing teaches self-control and calms the jumping nerves. Poise is attained by deliberately controlling the breath. We must teach self-control and establish self-confidence in as many ways as we can, so that it may react upon the speech.

Silent reading is a valuable exercise to train the child to obtain needed information from the printed page; but the child who has a speech defect must be definitely trained to read and talk orally as his speech mechanism does not rightly respond to stimuli, not particularly the brain, from without or from within. Knowledge must come first. Too often the stutterer tries to express an obscure thought. The oral organs begin to work before there is a clear, definite knowledge of what he wants to say. Pause is usually passed over as unimportant. Reading in concert should be given as class work for here he begins to realize that his oral organs can respond easily. Attention should here be directed to knowledge, poise of body, pause, rhythm, modulation, inflection more by force of example than by precept—as the teacher should read with the class. Speed is reduced, vowels are enlarged, articulation is less slovenly, continuity of voice proceeds better, and all of these results come without too much emotion. Then individual reading should immediately follow and criticism given which is constructive, not destructive. This may be followed by dramatization. Appeal here to the visualization processes brings less stilted action and a real co-ordination of the mental and physical activities.

By all this training, we shall find a very perceptible improvement; first, in the general poise; second, in a better control of the reading processes; third, in less emotional disturbance during speech, better speech in conversation, recitation and speech habit. Confidence is restored bit by bit and the violent speech reactions are laid aside. This is a long process. The child must be made over into a new being who views the world from an entirely different standpoint—but it is worth while. From being a cross-grained, ill-tempered, self-centered

or nervous individual, he blossoms out and often becomes a rather normal, healthy child who does not even realize the processes through which he has passed.

Can this be systematized so that class instruction, as well as individual work, may be given? Yes. Will the results be as good? Yes, since the training requires time; and each pupil is capable of absorbing only a little at a time; and class work saves a great deal of repetition. This saves the teacher's strength; and since this work is so concentrating, and good teachers do not grow on every bush, we must make and not mar them.

Must there be individual work? Decidedly yes, for here the individual must learn to apply to himself the things which he particularly needs; find out the fears, the interests peculiar to the child, dig down and root out his egotism, his phobias, his lack of self-control. If the correction has not been entire, adjustment has at least been made, so that the child can face unusual situations with more equanimity, and realities in a more normal manner.

For this work in mental hygiene, let us make a careful case history, inquiring, so far as we may, into the home; the social life; the personal habits; the characteristics; the health, physical, mental, and moral condition of the family in general, and the child in particular. Let us gain a very clear idea of his present speech and physical condition, and those experiences in the past which might have left their mark upon the subconsciousness. Let us find what his attitude is toward other people, and his ideas of how they treat him. Let us rid him of every possible physical disability. Let us make a careful survey of his fears, his phobias, his preferences and aversions, his attempts, his successes, his failures—search both the conscious and the subconscious mind to find a cause for this mal-adaptation to life situations. We must readjust him to his environment, teach him self-control, establish self-confidence, and teach him how to meet new situations without a speech breakdown. Make him think of the other fellow—the message that must go forth through speech—forget himself. When the stutterer can do this, our objective has been attained.

Fear is the evil genius of most stutterers. Train them to face situations honestly and make the best of them. Train them to concentrate, to attend. What they lack is the conviction that they can talk well. Terman says, "Once thoroughly convince the stutterer that it is possible for him to speak without tripping, and he is well placed on the road to recovery. Let him be taught not to strike, not to jeer, not to follow, not to give in, even though others do. Let him learn to take pride in being his own man, and not a puppet in the hands of others."

Detroit improves the speech of the stammerer through the following activities training in the fundamental principles of normal speech; physical education (massage, relaxation, poise, development of the fundamental muscle movements); voice training; visualization; phonetics, particularly the attack upon consonants; mental hygiene (talks with students); reading; spontaneous speech.

A STUDY OF ALLERGY IN RELATION TO STUTTERING

ROBERT E. CARD, M.A.
The Lewis Institute,
Detroit, Mich.

In former years the research worker sought the one hidden cause for stuttering. He thought this discovery would lead to a panacea which could be used for the cure of all stuttering. It is now generally accepted that most cases of stuttering result from a multiplicity of causes. One factor which has been given very little attention is that of nutrition, or the stutterer's diet.

In the first place it is necessary to recognize the fundamental law that the development or growth of an organism and its efficient functioning depends upon adequate nourishment. It is then most reasonable to conclude that speech, the most complex of the developing functions of the organism, cannot develop to the point of proficiency without proper nutrition.

It is important, also, to study this problem from the point of view of the Time-Place-Frame of Development,[1, 2] according to which "adequate nutrition" involves not only the proper chemical constituents and quantity of food, but also the Time and Place of its administration according to the growth gradient framework of the developing organism. It stands to reason that a failure to comply with the nutritional requirements in the growth processes involved in speech will be likely to result in some type of fractional speech.

Although stuttering usually is not classified as "fractional speech," because there are times when the stutterer can speak very fluently, the missing fraction very logically may be the incompleted development in the control mechanism of neuro-glandular-muscular activity.

If the growing structures of the organism have been sufficiently nourished, these control mechanisms will have developed so they control the speech processes adequately under all conditions. If not, there will be a lack of efficiency in the control determinants and hence in speech.

There are cases of stuttering which seem to have had adequate nourishment both as to quantity and chemical constituents. If such is the case, there must be a condition whereby the same food properties will not have the same effect upon the stutterer as upon the non-stutterer. Such a condition does exist and is present in certain people whom we speak of as being allergic or hypersensitive.

[1]The bearing these developmental processes on methods of speech correction has been expounded by Dr. C. L. Meader and Dr. John H. Muyskens, of the Department of Biolinguistics at the University of Michigan. It has proved valuable in the treatment of speech cases at the University Speech Clinic which is under the direction of Dr. Muyskens.

[2]Meader, C. L. "New Emphasis in Speech Rehabilitation." *The Quarterly Journal of Speech*, Vol. XXIV, 1938, pp. 233–238.

As is well known, allergy is a term used to express a condition in which a person reacts toward a foreign substance in quite a different way from that of a normal person. It is sometimes said that a person has an idiosyncracy or is hypersensitive toward a substance. We all are quite familiar with some of the more common symptoms such as hay-fever, asthma, eczema, and urticaria. The physician and laboratory technician agree that there is yet much to be discovered about the phenomenon, because of its complexities.

In the Time-Place-Frame of development, this condition of allergy may appear at a very early embryological stage, and because it may be inherited, it may even develop, or be present at the time of conception. According to Bray, "Hypersensitiveness in its clinical applications would appear to be possible of transmission from parents to offspring in one of two modes, either through the germ plasm or through the placenta."[3]

It can readily be acknowledged that certain types of reactions, should they occur in the embryo or fetus, could do considerable damage to the growing tissue. The same is true even after birth in the growing child. It seems most reasonable to believe, however, that the more free a developing organism is from any chemical upset, the more perfect will be its development and function as it proceeds toward maturity.

The question should be brought up here as to whether the cause or causes of stuttering were consummated during the period of unfolding and thus brought about a permanent weakness or inadequacy in the control mechanisms as mentioned above; or, as others have believed, the cause or causes are but a condition of metabolic imbalance during this period or later and which may be righted at any time during the life span of the stutterer. Those who have been studying stuttering from this latter point of view have only been able to discover so far that whatever abnormalities the stutterers may have, may also be found in non-stutterers.

Considerable work of merit already done by Kopp, Stratton, Starr, and others in the field of speech has failed to bring to light a definite metabolic imbalance as being the cause of stuttering. Let us hope that such a discovery will be made. It undoubtedly would not only help solve the riddle of stuttering, but of allergic reactions and many other maladies as well.

However, inasmuch as an allergic individual may have become allergic in utero or at a much later stage, allergic conditions could be the possible cause of stuttering in either case, through impairment of the control mechanisms which may in themselves have had full functional development, in a condition of imbalance.

While making a careful study of the general problems of stuttering and of allergy, a possible relationship occurred to me as a result of observing very striking similarities between the etiology and the manifestations of these two phenomena. The following are some of these very definite characteristic likenesses:

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[3]Bray, G. W. "Recent Advances in Allergy." P. Blakeston's Son & Co., 1936, p. 77.

1. The vagus or tenth cranial nerve plays a very important part in the speech process, and also is the nerve which is greatly concerned with the allergic reaction manifested particularly in bronchial asthma. Similarly other nerves that are involved in the allergic spasms of sneezing and coughing are also involved in the stuttering spasms.

2. There is ample evidence that certain types of epilepsy may be caused by sensitization, and have been cured by dietary control. Stuttering has very often been likened in its fundamental nature to epilepsy.

3. Many doctors whose practice include the treatment of allergic patients have observed that the first attack of asthma, hives, urticaria or other allergic reactions may follow an emotional upset. It is found that food or epidermals may bring on the attack, but in following a case it is noticed that usually a severe emotional disturbance will bring on the attack. Further analysis shows that the emotional disturbance was the precipitating factor and back of it all is foods, epidermals and substances to which the patient is sensitive. Still further study shows that it is impossible to analyze one's emotional field but one can usually change the entire color of the field by removing the sensitizing factors. In other words, the untreated allergic patient is usually irritable, easily depressed, easily upset emotionally. In fact he may be so upset that he is incoherent, may have such an advanced stage of stuttering that he can't talk. It is remarkable how most of this can be entirely removed by corrective diet and incidentally the entire emotional field has been changed.

Many of the old teachings of stuttering and its causes have been that it generally follows some emotional upset. In this respect it is no different from asthma. As these cases are followed it is observed that any emotional disturbance makes the stuttering much worse just as in asthma. As the case advances it becomes more and more irritable just as in asthma. In either case as they advance the nervous irritability becomes the most serious part of the clinical picture as time goes on. The end picture is that both types eventually become "nervous wrecks." It is known that this can be removed in asthma patients by diet. It seems only reasonable to suppose the same of these factors when applied to stuttering.

4. In a study made by Robert West in collaboration with Severina Nelson and Mildred Berry, on the hypothesis that stuttering is inherited, it was determined that stutterers have six stuttering ancestors as compared to one of the non-stutterer. They state further, "Would it not be reasonable, in view of what we have seen thus far, to say that apparently the thing that is transmitted from generation to generation is not stuttering but the tendency to stutter? Doubtless there are precipitating factors that cause the stuttering to appear when they are present in a given case, and cause it to remain as a hidden tendency when they are absent. Thus not all who have inherited the tendency to stutter actually stutter. Not all who can transmit this tendency to their offspring stutter themselves. Hence it is possible that in a given case only a slight tendency to stutter is inherited, but that the presence of strong precipitating factors will cause the stuttering to appear. In such a case the etiological significance of heredity would be minimal and that of environment maximal."[4,5]

It has been definitely determined that allergy is inherited and the above picture of the probability of stuttering being inherited is practically identical to that which would be found in a similar study regarding the inheritance of allergy.

5. Some stutterers are of the same general constitutional type as the allergic or exudative diathetic.

6. The native intelligence of the stutterer corresponds to that of the non-stutterer. Many superior children and adults stutter. In general, allergic individuals rank high in intelligence.

7. The onset of stuttering spasms in women are usually much more easily precipitated during the menstrual period and it is also true that the severity of allergic reactions are more severe in women during this same period.

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[4]West, R., Nelson, S., Berry, M. "The Heredity of Stuttering." *The Quarterly Journal of Speech*, Vol. XXV, No. 1, February, 1939, pp. 27–28.

[5]Blacker, C. P. *Chances of a Morbid Inheritance.* H. K. Lewis & Co., Ltd., London, 1934, pp. 37–38.

8. Allergic symptoms are usually relieved by injections of adrenalin and it has been observed by Dr. John H. Muyskens that in certain cases stuttering spasms are relieved by injections of adrenalin in the second phase of reaction in which the blood pressure falls.

The observation of many such evidences of similarity between asthma in particular and stuttering resulted in further interest and study. The author presented his hypotheses that stuttering spasms have a background of allergy, to Dr. John P. Parsons. Dr. Parsons saw enough merit in the observations to offer his advice and to furnish the antigens for this scientific study. Dr. W. W. Wellman also assisting in making the tests.

This research conducted in 1933 at the Lewis Institute suggests that all stutterers are allergic, and that this condition may be an important and basic contributory cause of stuttering.

Experimental Procedure

A case history was made. Its purpose was to determine the relative number of stutterers in whom conditions symptomatic of allergic reactions were present in the stutterer himself or in his family. Percentages warranted proceeding further and intradermal tests were made on a group of forty stutterers.

These tests were made with antigens with a tuberculin syringe and a 27 gauge 5/8 inch hypodermic needle. The positive reactions were recorded as to degree by the arbitrary classification of negative, 1 plus, 2 plus, 3 plus, and 4 plus.

Antigens were available for making tests of the following list of foods, pollens and epidermal proteins.

Beef	Asparagus	Apples	Chocolate
Chicken	Beets	Bananas	Coffee
Codfish	Lima Beans	Cucumber	Egg white
English walnuts	Oatmeal	Dates	Egg yolk
Herring	Onions	Grapefruit	Goose feathers
Lake Trout	Potatoes	Oranges	Orris root
Milk (cows)	Rice	Peaches	Rag weed
Perch	Spinach	Pineapple	Tea
Pork	Turnip	Strawberries	Timothy
Salmon	Yellow string beans	Tomatoes	Tobacco

The selection of the antigens for testing for allergic reactions in each case was confined as closely as possible to foods most common to the stutterer's diet. When the history suggested regular contact with certain pollens and epidermal proteins, tests for these were included whenever possible.

The white blood count was made with a standard hemocytometer, and the differential blood count was made by staining with Wright's Blood stain. The percentage of hemoglobin was estimated by comparing the color of blood with standard color comparators. (Talquist Method.)

Data and Discussion

(A) *Case Histories.*

Of the one hundred four case histories of stutterers and their families, one hundred and two indicated that allergic symptoms had

been noticed in themselves or in some member of the family. This was true in one or more of the forms listed. The two whose cases did not report positively, when tested intradermally, gave very positive reactions. Stutterers, like the asthmatic and other type of allergic patient, show a positive family history of allergy which in this series ran better than 98%. It would undoubtedly be 100% if this study could be perfected.

It has been observed by Rowe that the percentage which shows a positive family history is low because of the impossibility of obtaining a true interpretation and record of the various illnesses of the different relatives of the patient for three generations.

Of the one hundred and four histories obtained from non-stutterers at random, 62% indicated an heredity of allergy. From the extensive list of symptoms in the questionnaire, it would compare favorably with the findings of Rowe which he gives as follows:

	Per cent
People with positive family or personal records of probable allergy	58
People with questionable evidence	16
People with negative evidence	26

In 1934, Jimenez, in a study of 6935 University of Michigan students, obtained the following data:

	Per cent
History of probable sensitization	35
History of probable potential allergy	20
History of probable nasal and bronchial allergy	11.9
History of probable good family history of allergy	22.27
History of probable food allergy with no family history of allergy	19.19

A more recent study by Jimenez (1938) with more figures available indicate that approximately 53% of the student body need definite consideration from the standpoint of allergy.

These findings, however, are very high compared to Balyeat who stated in 1930: "About seven per cent of the population of the United States are sufferers from hay fever, asthma, or allied conditions. These allied conditions spoken of are hives, eczema, migrane, and probably some cases of mucous colitis and epilepsy."[6] It is believed that Balyeat and others who have figured such low percentages did so because of failing to include many symptoms in this category.

(B) *Intradermal Tests* (Forty stutterers).

The author would have liked to have tested each patient to several hundred antigens, but felt this was impractical because of physical limitations. He felt he might better do a smaller number, do it carefully and be sure it was done in all cases.

All of the forty stutterers showed reactions to the intradermal tests made. A total of 726 tests were administered. Of this total, 352 or 48.5% showed positive reactions in the following degrees of severity:

(1) 85 reactions or 11.7% of total tests rated 4 plus.
(2) 105 reactions or 14.5% of total tests rated 3 plus.
(3) 85 reactions or 11.7% of total tests rated 2 plus.
(4) 77 reactions or 10.6% of total tests rated 1 plus.

[6]Balyeat, R. M. *Allergic Diseases.* F. A. Davis Co., Philadelphia, 1930, p. 15.

Of forty tested, the number of stutterers reacting to each degree of severity in one or more tests are as follows:

4 plus	3 plus	2 plus	1 plus
33	36	33	32

It was quite noticeable that the severity of the stuttering was usually in proportion to the percentage of the reactions and/or their severity.

In discussing positive skin reactions to demonstrate good sensitization, Schloss stated in 1915 that the main difficulty in the diagnosis of food allergy is obtaining positive reactions. Rowe says that the frequency of negative reactions of food causing active symptoms is becoming generally recognized, and "I have long known that satisfactory reactions are absent to less than 50% of clinically reacting foods."[7]

In spite of the small number and low degree of reaction which might be expected from these observations, the results of these intradermal tests seem remarkably high and would indicate that the stutterers might even be more hypersensitive than the tests reveal. On the other hand it has also been observed that there are positive skin reactions for which no clinical symptoms can be observed.

Since I announced this research when giving a talk at a meeting of The Schoolmasters Club in the spring of 1938, stutterers have been tested for allergic reactions at the University of Michigan Health Clinic and Dr. Muyskens reports that to date their findings substantiate the author's findings as outlined in this article.

(C) *Blood Count.*

The blood count of those tested seemed to come within the normal range. Twenty cases were tested for white blood count which varied from 3700 to 10,500. The esoinophil count varied from 0 to 5 per cent. The percentage of lymphocytes including both large and small varied between 9 and 41 (average 26.9 per cent). This also was not as high as might be expected in comparison with most allergic patients.

Practically all stutterers have periods of various durations in which they speak with very little or no stuttering. This indicates that if stuttering is induced by allergic reactions, blood counts taken just before, during, or immediately after stuttering attacks would differ from those taken during periods of relief. Consequently, in order to determine more accurately the blood count as an indication of the relation between allergic reactions and stuttering, a system of checking and severity of stuttering in relation to the time of taking blood counts should be established.

(D) *Foods to Which the Stutterer is Sensitive.*

The largest number of food protein tests showed the most severe reactions in the following order of frequency: pork, fish, nuts, spinach, tomatoes, oranges, strawberries, and milk (cow's). There are other foods, however, not used in this test to which a greater proportion of stutterers might have severe reactions. It should be borne in

[7]Rowe, A. H. *Clinical Allergy.* Lee & Febiger, Philadelphia, 1937, p. 48.

mind that this test was to determine the sensitivity of stutterers to food proteins and not to determine the foods to which the stutterers as a group are most sensitive. It should also be remembered that the food to which one allergic person is the most sensitive may have no effect upon another equally allergic person. However, allergic people generally are more apt to be sensitive to certain foods than they are to others. It seems reasonable, therefore, that a general elimination diet might be prepared for stutterers as a class.

(E) *Alleviation of Stuttering by Elimination Diets.*

Elimination diets by empirical tests apparently have greatly reduced the stuttering spasms in two adult cases. Both had stuttered for over thirty years.

Tests through eliminating allergens from the diet of stuttering children have not been given sufficient trial to evaluate the effect on speech.

To determine accurately the foods to which the individual stutterer is sensitive is a difficult problem because of the great variety of foods consumed. It is also true that a person may be sensitive to a food allergen and still show a negative reaction to a scratch skin or intradermal test.

Summary

Case histories indicate that all stutterers are allergic. The average severity to allergic reactions indicate that stutterers as a group are more sensitive than the average group.

There are many definite similarities between allergy and stuttering.

The severity of stuttering seems to be in direct proportion to the percentage of reactions and/or their severity to tests for sensitivity.

Blood tests for comparison should be taken during a period when the stutterer is most free from stuttering and again when the stuttering spasms are most severe and frequent.

Because it is more difficult to obtain knowledge of all the substances to which one is allergic and the degree of sensitivity to each one, an effective elimination diet is not easily determined. However, allergic people generally are more apt to be sensitive to certain foods than they are to others.

Elimination diets have in some cases definitely reduced the nervous irritability and stuttering spasms of stutterers.

Conclusion

The fact that nutrition is a fundamental determinant of energy, storage, release, and distribution in the neuro-muscular glandular activities of the organism, as well as the basis of the phenomena of allergy, makes it logical to conclude that allergic conditions could be a possible cause of stuttering either because of impairment of the control mechanisms in the embryonic or later developmental stages, or because of a condition of functional imbalance in otherwise adequately developed structures. Because of this fact the Time-Place-Frame theory is an important point of view to keep in mind in attacking the problem.

Similarities between the factors of stuttering and asthmatic reactions; the positive histories of allergy in the stutterer's families; the fact that every stutterer tested showed a marked degree of sensitivity; and the degree of stuttering seemed to be in direct proportion to the severity of reaction to the intradermal tests—all of the above definitely indicates the likelihood of stuttering being in such cases a manifestation of allergic reaction.

It is believed that this preliminary study reveals sufficient evidence to warrant further studies of these relationships.

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A STUDY OF LATERALITY OF STUTTERERS AND NORMAL SPEAKERS

BRYNG BRYNGELSON, Ph.D.
Director of Speech Clinic, University of Minnesota

In 1937 the author, assisted by Bernice Rutherford, completed a comparative study of laterality of stutterers and non-stutterers. The factors under consideration were: present handedness, shifting from left to right in handedness, history of left-handedness in the family, and history of stuttering in the family. The cases presented in that study numbered 74 in each group, ranging in age from 4 to 16 years. The results were published in the *Journal of Speech Disorders* for March, 1937. (1)

The object of the present study was to ascertain the differences, if any, in the laterality of a group of stutterers and controls at a higher age level than those studied in the earlier research. Subjects ranging in age from 17 to 31 years were used in this study. The experimental group of 78 stutterers were matched with a control group of 78 normal speakers. The two groups were paired as in the former study according to chronological age, sex, mental age, and social status. There were 29 males and 49 females in each group.

Both the experimental and the control groups were studied clinically. Similar case history questions and clinical tests were administered. In order to lend diversification and increased reliability to the study, five Speech Pathology majors assisted the author in the administration of the case history and the clinical tests for sidedness.

The table below presents the data on the four most important items studied.

TABLE I

(29 males; 49 females)

	78 Normal Speakers	78 Stutterers
Present handedness:		
Right	94%	69%
Left	6%	2%
Ambidextrous	0%	29%
Shifted left to right	1%	58%
Hist. left-handed in family	42%	49%[1]
Hist. stuttering in family	6%	54%[1]

[1]Both immediate and remote history of left-handedness and stuttering are included.

There are two main differences to be noted in these two groups; namely, shift in handedness and ambidexterity. A considerably greater number of the experimental group have experienced a shift in handedness, and a larger per cent of the experimental group than in the control group were ambidextrous. The percentages noted differ significantly when treated statistically.

It is also interesting to note that the experimental group had 50 per cent more enuretics in their number than did the control group. The enuresis persisted beyond school age. I mention this item because a few workers in the field have pointed out a possible relationship between an unstable nervous system and enuresis. It is possible to conceive of a weakened control of an organ, bilaterally innervated, when the higher neurologic levels are disturbed as might be the case in the central disturbance we call dysphemia, of which stuttering is a peripheral symptom. This study also revealed the fact that fewer of the experimental group had experienced significant childhood diseases. Many workers have attributed stuttering to severe childhood diseases. The author does not deny that febrile diseases may play a significant part in the etiology of stuttering. There are not a few patients whose history reveals no other factor incident to the onset of stuttering. However, in this study 28% of the control group had had severe childhood diseases while in the experimental group only 12% indicated such in their history.

The two main factors of interest, the high incidence of shift of handedness and ambidexterity in the stutterer, seem to appear rather consistently in other groups of stutterers studied at the University of Minnesota Speech Clinic. Recently compiled data (only half of which has been published to date) (2) on 1421 patients studied by the author indicates shifting of handedness in 73% of the cases, and 53% were ambidextrous.

For purposes of viewing the study of laterality in relation to stuttering over groups ranging in age from 4 to 31 years, the following table combines both the groups already published and the present study.

TABLE II

(62 males, 90 females; age 4–31 years)[2]

	152 Normal Speakers	152 Stutterers
Present handedness:		
Right	83%	63%
Left	11%	3%[3]
Ambidextrous	4%	34%[3]
Shifted left to right	5%	61%
Hist. of left-handedness in family	53%	64%
Hist. of stuttering in family	13%	51%

[2]The data is on file at the University of Minnesota Speech Clinic.

[3]When the statistical significance of these percentages is computed on the basis of the Edgerton-Paterson (3) tables for determining the significance of percentage differences we find the difference over the standard deviation of the difference is 7.14 for ambidexterity and 12.72 for the shifting of handedness.

Discussion

Many laboratory studies by Travis (3) have previously pointed to the fact that stutterers as a rule fall into the mixed dominance group. In other words, the central mechanism of a stutterer appears to be in a state of ambilaterality, neither the right nor the left cerebral hemisphere exercises a dominant lead control over the other. In normal speech it is postulated that because of a dominant hemispherical control the midline speech structures function synchronously. In a stutterer no such cortical superiority of function seems to obtain. This may be due to the fact that for one cause or another there does not exist in the stutterer a condition of strict one-sidedness, a condition which appears to obtain to a larger degree in normal speakers. There is further evidence, clinically, in this study that stutterers are not as definitely one-sided as normal speakers. When footedness, eyedness, and handedness (unimanuals and bimanuals) were correlated in both groups of 152 cases each, only 19% of the stutterers were strictly one-sided, while 51% of the non-stutterers were one-sided in the three correlated motor functions, the critical ratio being 6.15.

Another factor of interest is that, whether the original study of 74 cases is considered separately or in combination with the second study of 78 cases herein reported, there does not appear to be as great a difference as was formerly thought to exist in the stuttering and non-stuttering groups in the matter of family history of left-handedness. The experimental group of 152 cases had 11% more left-handed relatives than did the control group. The critical ratio is 1.93, which is not statistically significant. It should be noted, however, that the control group happened to have in it an unusually large number of left-handed individuals—a fact which has not, as far as I know, been true in studies of the normal speaking populations reporting only 25% to 30% of left-handedness in the family stock. It is obvious that if one studies an inherited trait, as left-handedness, that the history of left-handedness would be higher in a group in which there were a greater number of left-handed subjects than in a group composed entirely of right-handed subjects. This same fact of differentiation is also indicated in the matter of eyedness. Thirty-four per cent of the 152 controls were left-eyed, while 51% of the experimental group were left-eyed. In studies of right-handed groups of normal speaking subjects, approximately 25% have been found to be left-eyed. Fifty-one per cent of the 152 stutterers were left-eyed, which places them in the left-handed group as far as sighting preference is concerned.

Stutterers and non-stutters are definitely differentiated on the item of family history of stuttering. The combined studies of 152 cases reveal 4 times greater stuttering history in the experimental group than in the control group (c. r. 7.76). West (4), Berry (5), and Wepman (6) would interpret these facts as evidence for the inheritance of stuttering. To accept that thesis in toto the author is

unable to do at the present time. The possibility is granted that the central mechanism of bi-hemispherical antagonism in neural discharge is present at birth in some children causing peripheral myospasms to obtain on the speech mechanism when the child first attempts speech communication. But to say that this is the condition in all children who stutter, and consequently there is nothing to do about it, the author cannot at present accept. We may some day be able to distinguish types of stuttering, one type represented by a state of central ambilaterality present at birth as a genic trait, and another type represented by the same central condition established as the result of various environmental and developmental factors including the alteration of native sidedness. At present, however, speech pathologists note only two symptoms common to all dysphemic patients; namely, tonic and clonic spasms. In the research presented here, the author has called attention to three additional factors which appear to differentiate stutterers from normal speakers; namely, the degree of peripheral ambidexterity, the percentage of handedness shifting, and the degree of strict one-sidedness.

Further research on this intriguing malady is warranted. There is no reason to believe that, because the search for the etiology of stuttering has obtained for nearly 2,000 years this is the age in which speech pathologists will find its ultimate and final truth.

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Le VIIème Congrès de la SOCIETE FRANCAISE DE PHONIATRIE aura lieu le Mardi 17 Octobre 1939, à 9 heures précises, dans le grand amphithéâtre de la Faculté de Médicine de Paris.

Rapport présenté: DR. PICHON and MME BOREL-MAISONNY: "L'aphasie et les troubles psychogènes du langage."

Secrétaire Général
DR. TARNEAUD
27 Avenue de la Grande Armée
PARIS (16°).

A STUDY OF THE ORAL RECITATION PROBLEMS OF STUTTERERS*

THELMA A. KNUDSON
Director, Speech Correction
Central High School, South Bend, Indiana

This study was made in order to determine the various types of methods used by teachers in handling stutterers in the oral recitation situation and to determine the effect of these methods upon the stutterers. The study was prompted by the need for more definite knowledge concerning the techniques that should be used by teachers in handling stutterers in the classroom.

I. Subjects and Procedure

For this survey seventy-two stutterers and fifty teachers served as subjects. The stutterers included sixty boys and twelve girls, thirty-six of whom were from the public schools of South Bend, Indiana. The remaining stutterers included ten from the Speech Clinic at the State University of Iowa, nine from Purdue University, and seventeen from the East Chicago, Indiana, public schools. The age range extended from nine to twenty-seven years with an average of fourteen years. The fifty teachers, ten men and forty women, from grade 1 through senior high school, were chosen from among those who had had stutterers in their classes. Information was secured from the stutterers by means of personal interviews, supplemented by a questionnaire. A second questionnaire was used to secure information from the teachers.

II. Results

The responses made by the seventy-two stutterers concerning methods used by their teachers in connection with the oral recitation show that twenty-nine of these stutterers made use of written work to compensate for their shortcomings in oral recitation, while forty-one did no extra written work. Sixty-two out of seventy-two felt that they made poorer oral recitations than they were intellectually able to make. The remaining ten were mild cases and their stuttering had not hampered them greatly in oral recitation. Approximately fifty

*More complete data and materials pertinent to this study are in the manuscript copy of this investigation on file in the library of the University of Iowa. The study was made under the direction of Dr. Wendell Johnson.

per cent admitted having given the wrong answer or having said, "I don't know," in order to avoid the speaking situation. For instance one stutterer says, "I found it easier to say 'I don't know,' than to answer although I often knew as much about the subject as anyone else in class. I still find it easier to say 'I don't know' than anything else in the English language.

Another stutterer gives this experience, "One professor, who was a very good friend of mine, used to call on me once in a while but I always smiled and answered, 'I don't know,' or just shrugged my shoulders and he casually passed on to someone else. One day he asked me, 'When did Columbus discover America?' I answered that I didn't know, and he got a good laugh out of it and I did too, but if anyone else had done that I would have dropped the course." Thirty-one, or almost fifty per cent, reported having been retarded in school because of their stuttering, but only eleven believed that their teachers were responsible for their retardation.

Six of the seventy-two interviewed admitted having played truant because of their stuttering. In each of these cases it was in order to avoid giving an oral recitation or report. A number of others admitted that they would like to have played truant but were afraid of the consequences.

Only six stutterers felt that they should be excused from oral recitations entirely. The remaining sixty-four believed that some oral work was advisable, since it would tend to give them more control over the speaking situation and makes them feel less inferior. The majority, however, felt that some consideration should be shown them and that they should be excused from recitations that were too long or too difficult to undertake. Most of them thought that the most satisfactory policy would be to let them recite when they volunteered to do so.

Over fifty per cent of the stutterers reported that none of the various methods used by the teachers was particularly helpful. A kind, friendly, jolly disposition was the type of personality most of them considered essential in a teacher. Forty-three per cent replied that they had teachers who did not know that they stuttered. Some of these were mild cases, and the more serious ones concealed their handicap from their teachers by not reciting and by remaining much in the background. However that may be, the indications are that estimates of the incidence of stuttering are probably inaccurate when based upon teachers' judgments.

A survey of the teachers' responses show that the most common reactions that teachers have towards stutterers are sympathy and pity.

Forty out of fifty teachers definitely considered stuttering beyond their ability to cope with along therapeutic lines.

Thirty-two different methods were used by the fifty teachers in handling stutterers in classroom, indicating that, as a general rule, teachers are neither very definite nor very consistent in their policies

regarding stuttering pupils. Nineteen different methods were advocated by different teachers as being the most effective. The largest number of teachers maintained that building up confidence and making the stutterer feel at ease are the most effective methods. It should be pointed out, however, that these are really not methods, but aims.

Sixteen different causes were given for stuttering, indicating a wide variance of opinion among teachers upon this subject. The largest group of teachers, comprising fifty per cent, believed it to be caused by some form of "nervousness." Among other reasons given were lack of confidence, habit, imitation, lack of control of throat muscles, and malformation of the speech organs. Since a teacher is inclined to treat a stuttering case according to her idea of the cause, one can see the serious results that might ensue through a lack of knowledge upon this subject.

III. Stutterers' Reactions

The following quotations are taken from the questionnaire responses of various stutterers in this study and are given as representative of the group reactions. They furnish further insight into the problems of stutterers in the classroom.

"I remember two teachers who were far from being just, mostly, I believe, because they were so impatient. The first teacher that I remember as being impatient was the fifth grade teacher who was generally feared about the school because she was too exacting and intolerant. She acted toward my stuttering as though I was generally incompetent and slow.

"A teacher that was given to constant sarcasm and cutting remarks made it twice as hard for me. I felt that every time I would make a recitation she would likely make some cutting remark if the answer was wrong, so rather than take a chance on such criticism I would say as little as possible in her class. One of the greatest drawbacks is a teacher who is always trying to help by supplying the answer before you can say it, if you start to stutter or hesitate. They mean well but many times I have wondered why they ask me in the first place if they are going to give the answer as soon as I stand up."

"Only one teacher made me feel inferior to the class and after that I felt that way in every class. That was the teacher who forced me to give an oral report in geography. The report was well written, but a flop orally and I felt it a long time after. A few teachers forced me to recite, but I avoided this by playing dumb."

"I found that I could get along much better with a teacher that had patience and would give me more time to answer questions than one that made me sit down because she couldn't waste time."

"If a teacher seems interested in what you are saying, that helps more than anything else. If her attitude is indifferent, I would never bother saying any more than I had to at any time and would never recite unless called upon."

"I remember one teacher that called on someone else when I was trying to recite. This upset me to a great extent and gave me the further impression that my stuttering was not only a burden to myself but also to other people."

"In geometry the teacher had a system of recitation that was excellent for me. Every day about a third of the class would put problems on the board and then someone else would explain them. I usually got to put one on the board and that showed I knew it. Then someone else would explain it and I didn't have to do any talking. Naturally I liked geometry very much."

"Most of the time I enjoy going to school, for I like to be among people but when I have to give a speech or an oral theme I dread school and wish I could die so I wouldn't have to give my speech. Some days I feel all tightened up and I feel just can't get any words out and I'm tempted to stay out of school, but my mother makes me go and I have to face it."

"In college my stuttering continued to color more and more my school life. The professor of my freshman chemistry class, a blunt, direct, loud-speaking old fellow, insisted that I recite in his discussion section. So I recited now and then throughout the year, but my speech did not improve and I certainly felt no better about it. These recitations embarrassed me, and I felt more or less separated from the other members of the class. I never entered into their light conversations, and didn't even speak to them for a long time. The only thing that saved me from losing completely my self-respect was my ability to stutter out the correct answer to most of the questions. This method of treating stutterers with its inevitable stunting of one's social growth, however, is a precarious way in which to foster scholarship."

"I think the best thing a teacher could do, if he had the time or interest, would be to talk to the student after school hours and try and change his attitude towards his speech and call on him once in a while and allow him to talk as long as the stutterer felt he wanted to. The stutterer would always feel that he had at least one person in the room who was with him in his attempt to talk. But this would only help if the teacher had been able to change the stutterer's attitude to some extent."

"I have mentioned but a few of the outstanding incidents which illustrate the intimate relationship existing between a stutterer's school life and his stuttering. Many more could be listed if I could remember them. At any rate, I hope that these few will impress upon the teachers the responsible position which they hold being where they can adjust the stutterer to his stuttering. Innumerable moments of misery could be turned into moments of happiness did but one teacher know how to treat properly one stutterer."

IV. Conclusions

It would appear that to a significant degree the attitude of stutterers toward school and education generally are determined by their experience in oral recitation. On the whole, these experiences are not conducive to attitudes that facilitate learning. Moreover, the experiences of stutterers in connection with oral recitation appear frequently to be more detrimental than beneficial as far as general personality development and speech development are concerned.

These facts constitute problems for the school staff and especially for the classroom teacher, since she is the one who most directly determines the nature of the experience which the stutterer undergoes in the oral recitation situation.

The findings of the present study indicate that a large majority of the classroom teachers interviewed consider themselves inadequately trained for meeting these somewhat unusual and subtle problems. This seems true both with regard to their knowledge of stuttering and their familiarity with principles of dealing with the adjustment problems occasioned by stutterlng. Not only do the teachers consider themselves to be inadequately trained with regard to this special problem, but also the stuttering pupils themselves express the belief that the teachers' methods and policies are often unsatisfactory and frequently unwise. This implies that in so far as stuttering resembles other behavioristic deviations found among

school children, classroom teachers as represented by the ones interviewed in this study have not had adequate opportunity to become trained to handle classroom problems associated with such deviations. It is reasonable to recommend a re-evaluation of current teacher training facilities and policies in so far as this study indicates the need for such re-evaluation.

V. Recommendations

Certain positive recommendations may be made, at least tentatively, on the basis of the present study.

First, some type of special consideration should be given the stuttering pupil in regard to oral recitation. The plan probably most desirable is to call upon the stutterer to recite only when he volunteers to do so.

Second, the stutterer should not be hurried. He should neither be interrupted in his efforts at speaking, nor should an attempt be made to say the words for him. Irritation, indifference, or an expression of boredom on the part of the teacher creates a decidedly unfavorable reaction in the stutterer.

Third, such an atmosphere should be created on the part of the other members of the class as will make the stutterer feel that he is not excluded from, nor inferior to, the rest of the group.

Fourth, it is usually desirable to demand extra written work of the stutterer to the extent that he is excused from oral recitation.

Fifth, it is especially desirable to develop a sincere, friendly interest in the stuttering pupil, to gain his point of view, and to establish his confidence through personal conferences concerning his problems and how he may best meet them.

Sixth, it is desirable to prepare the stutterer emotionally and intellectually to meet as many speech situations as possible. He should in no case, however, be forced into them, but rather the instructor should strive to create a desire in the stutterer to take part in oral recitations.

It is not to be expected that the classroom teacher know how to treat stuttering clinically. Rather, emphasis should be placed upon the desirability of the classroom teacher's knowing how to train the stuttering pupil to develop such attitudes toward himself and his disorder as will facilitate his scholastic progress and general personality development. In a word, if the teacher cannot cure stuttering—and it is not reasonable to expect her to cure it—her job becomes that of teaching the child to live with his stuttering as gracefully and efficiently as possible.

BOOK REVIEWS

A PRACTICAL PROGRAM OF SPEECH CORRECTION. By H. J. Heltman (Syracuse University). *The American School Board Journal*, 90: 31–32, 90 (June) 1938.

We have long needed a paper written by a speech corrector for school administrators and published in a school administration journal. There was no one better able to write this than Professor Heltman, whose actual experience with this relationship has been of the broadest. To one as enthusiastic about this plan as I am, Heltman seems to have stated his point of view very conservatively. This paper will not only result in an advance to our profession, but should be required reading for every speech therapist, so that he can converse familiarly on the subject to school superintendents in their own language. I believe this to be one of the most important papers of the year. C. H. V.

SCIENCE AND SANITY. By Alfred Korzybski. Lancaster: *The Science Press*, pp. 798. $4.50.

Count Korzybski extends the field of speech correction beyond that of what is generally thought to be the limitation. In fact, he described a whole new syndrome of language disorders. He calls the book an introduction to non-Aristotelian systems and general semantics. The principal differences between general semantics and other psychological treatments of stuttering and language disorders are (1) the direct approach through extensionalization which cuts the treatment time down, and (2) the attack of both the cause and the symptom together. Former psychological methods were apt to treat the original cause which had long since ceased to operate, and when finished still have the symptom, such as stuttering speech, left. General semantics attempts to remove the original cause, the cause operating in the present, and the manifest symptom. At the beginning of each chapter are illustrations showing that the ideas to be presented in the chapter are not new, but have been pointed out before. These quotations are clearer if read after having finished the chapter. C. H. V.

THE PRONUNCIATION OF VOWEL SOUNDS, AN EVALUATION OF PRACTICE MATERIAL FOR COLLEGE FRESHMEN. By E. G. Kuhn. New York: Teachers College Publications, Columbia University (No. 757), pp. 86, 1938. $1.60.

Speech correctors have been divided in their opinions as to whether to use disconnected sentences or poetry for drill material in the amelioration of parathonoontalalia. Kuhn's research has attacked this problem as it pertains to late adolescents. Poetry was found to be as good as and even slightly better than disconnected sentences. C. H. V.

INTRODUCING SPEECH CORRECTION INTO A NEW SCHOOL SYSTEM

ROBERT MILISEN
Indiana University

The subject of introducing speech correction into a public school system is one of the most important matters confronting the members of the Speech Correction Association. The success of introducing a speech correction program is dependent upon many factors. However, most important of all is the avoidance of antagonistic feelings and reactions on the part of parents and school teachers. Many speech correction programs which otherwise would have been successful have failed because the speech correctionists were not cognizant of this fact.

The problem is first of all divided into two parts. First, the school system must be made ready to have a program introduced. In order to achieve this goal many procedures may be followed. Perhaps the most successful is the traveling clinic. As Dr. Strothers has told us this morning, the traveling clinic is a dramatic way of presenting the problem to the public. It often astounds people when they realize and can see for the first time a large group of handicapped children who are receiving no training. The traveling clinic may further stimulate interest by producing improvement in a number of cases. This is always very encouraging to a community and oftentimes receives widespread publicity in that community. The third contribution of the traveling clinic is adult education. This is achieved by means of demonstrations to parents and teachers and through newspaper publicity. The traveling clinic at this time is extremely important to the future success of speech correction. Some states have been carrying on a traveling clinic project, but most of them have not. If the clinic can be organized on a state-wide basis, speech correction can be moved ahead more rapidly and in a more unified fashion.

Let us now assume, however, that the school system is convinced that a speech correction program is necessary. The problem arises then how one can best develop a program for that school system. I feel that speech must be placed on an even basis with other studies such as reading, arithmetic, spelling, and so forth. We must realize that speech is the most valuable contact the child has with the outside world and that the speech of no child is perfect. In other words, all children need to work for speech improvement. This can be easily demonstrated to the school system by showing that forty or fifty percent of the children mispronounce common

words such as *get, just,* and *because.* Next there are children whose articulation is so slovenly that it is difficult to understand the child's speech. And then there are the voice problems presented by children who cannot make themselves heard. All of the children in the school can benefit by a speech improvement program; so this first year of the speech correction program should be devoted primarily to the establishing of speech improvement work throughout the school system. Ordinarily this improvement program should include all of the elementary grades starting with the kindergarten.

Speech improvement programs have been worked out which absorb from five to ten minutes of the school day and are so well worked out that the class room teacher is not required to do any additional work toward their preparation. Herein again lies a very important factor in avoiding antagonism. The class room teacher is already overburdened with duties. Anything that we do that adds to her heavy load will be ungraciously received, and co-operation will be poor. On the other hand, if the program is attenuated to the reading and spelling program so that all words used in the speech improvement project have been taught previously, the load would not be as heavy as it might seem. The entire stress may be placed upon speech and not upon learning to read the word. This, of course, makes the teacher's problem easier also. The games and drills should be clearly and distinctly described, thus taking more of the burden off of the teacher.

The reports that I have had from school systems which have inaugurated such a program indicate that, not only do the teachers enjoy the speech improvement work, but also the children react very favorably to it. The study is a new one, and it is different. Although it is not a relaxation period, yet owing to its disassociation with other academic subjects, it has the same effect. Children become more conscious of their speech and that of others around them, and as a result, not only does their own speech improve, but also we observe an improvement in their English work.

When we introduce speech improvement programs into the school systems, we eliminate all of the marginal defects which might cling on into adult life and thus permit the speech correction teacher to spend his time with only the severely handicapped children. The speech improvement program would take care of approximately eighty to ninety percent of the so-called defective speakers reported by Dr. Morris in his speech survey discussion in this morning's meeting. In relation to this paper, I wish to suggest that in my opinion a survey which places the number of speech defectives at forty-five or forty-six percent is more dangerous than helpful to speech correction. The reason for this is that the average superintendent will laugh in your face if you mention such an extremely high percentage of the children as being defective speakers and will tend to discount every truth about speech correction that you may state later on. Herein lies another source of antagonism. The reason for this, of course, is that according to such a survey

probably forty percent of the adults even in the educational world would be considered defective speakers; and superintendents ordinarily reason this way, "If I am a defective speaker and I have gotten as far as I have in the educational field, then speech disorders cannot surely be a very great handicap." A child should be considered a defective speaker only when his defect is severe enough to attract the attention of the layman and perhaps thus handicap him in social intercourse. One can see that all of the children in the school system need speech improvement work without necessarily calling them speech defectives. By using the same logic, forty to fifty percent of the children may be below the average in reading and need special help; yet we would not necessarily call all of them reading defectives. Unfortunately there is a certain stigma attached to the words "speech defective." Hence it is well to think carefully before applying the term to any individual.

Introducing Speech Improvement

It should be our earnest effort to place speech on the same par with other academic subjects before we introduce speech correction projects. If we are able to do this, then the children who are defective speakers according to anyone's judgment will not feel so terribly isolated and so different when they receive special help for their speech defects. Instead, when a teacher is prepared to help the child with his speech disorder, he will view this assistance in the same light that he would consider special help in arithmetic or reading or any of his other subjects. I feel that insuring good mental hygiene for the child is far more important than any speech correction or speech improvement work that might be achieved without development of good mental hygiene. This is especially true in light of the fact that a great percentage of the elementary school children who have speech disorders are aware of their disorders.

The problem of introducing speech correction is manifold, first because in our minds speech correction often takes on many attributes which are not primarily speech corrections, but rather are associated disorders. Hence special disorders in reading, in spelling, in hearing, and in locomotion may complicate the picture. In the remaining discussion, however, we will consider the problem principally from the standpoint of straight speech correction work.

Introducing Speech Correction

The next question arises as to the nature and size of the school system and the amount of state aid that can be obtained for this speech education and correction work.

The small town.—First, let us consider the problem from the point of view of the small town with a population ranging between ten and thirty thousand. A town of this size can easily afford work for one full-time speech correctionist, and under existing conditions more than one will be needed. However, let us consider this just the

beginning and be very thankful for the addition of one full-time staff member. The addition of even one teacher into a small school system does not appear to be an unbearable burden, and this staff member coupled with the speech improvement program should be able to offer fine assistance to the majority of the more severely handicapped children in the school. This teacher's time, as has been stated previously, should be spent primarily during the first year with the speech improvement program. However, if he has some free time, he might devote it to defective speakers in the higher grades. The justification for this is that the students in these grades will be graduated before any assistance can be offered to them. The second year he should be able to devote an increased amount of time to speech correction itself and less time to the speech improvement program. I would suggest that even in the second year of the program, rather than have the correctionist spend a great deal of time with a speech survey of all of the children in the school system, he should depend upon a general report of the number of speech defectives in each room from each classroom teacher. There are many weaknesses to such a procedure. However, the average teacher, after one year of speech improvement work, will have a much more intelligent perspective on speech defects and will give a much more accurate report than would the average teacher in our school systems today. Another justification will be that we should conserve as much as possible the time of this speech correction worker. The primary purpose of a survey is to locate the children who have defects, and the classroom teachers will be able to report more than enough of the cases who need help to take up the time of the speech correctionist. A survey might logically be conducted during possibly the third or fourth year. There are, however, many exceptions to such a program. If the speech correction teacher still has the job of selling the program to the superintendent and the school board, a speech survey might logically be conducted during the first year. The statistics showing the high prevalence of speech defects among the school population is a very powerful device if used properly.

The large town.—Introducing a speech correction program into a large town of 100,000 population or more oftentimes becomes a more difficult problem, the reason being that a town of 100,000 should have at least five or six speech correctionists if the defective speaking children in that school system are to be treated adequately. The introduction of a burden of five new teachers into a school system of that size oftentimes will be viewed as financially prohibitive and as a result will cause these school boards to refuse even to start with one special teacher. Children not served by this correctionist and their parents will often raise objections because they are not a part of the program, until the school system is forced to hire a large number of corrective teachers. Hence the superintendent will often refuse to start a program which he isn't sure he can finish.

It is for this type of school system that I recommend the following program: one well-trained correctionist should be employed whose primary functions will be (1) to inaugurate and supervise the speech improvement work, (2) to oversee the speech correction work, and (3) to offer therapeutic treatment to the more severely handicapped children in the school system.

At the same time that the correctionist is hired, one teacher should be chosen in each building to take charge of the immediate speech improvement and correction program in that building under the guidance of the speech correction supervisor. It should be a kindergarten or junior high school teacher, and speech correction should be considered a teaching subject, thus becoming a part of the routine duties. The qualities which this teacher should have are (1) an active interest in speech correction, (2) a background and understanding of psychology, and (3) a willingness to advance her educational background by taking speech correction courses from some accredited university and also to participate in school teacher meetings arranged for by the speech correction supervisor.

During the first year these untrained individuals should not attempt to do remedial speech correction work. Instead they should begin some courses of study in speech improvement and speech correction. Certainly the first course that should be considered would be a fundamentals course in the treatment of articulatory disorders. The courses to follow should be prescribed primarily by the speech correction supervisor. During the first year the junior high school teacher should be able to contribute to the organization of speech improvement work in her school system. During the second year she should be able to begin corrective work for articulatory defective children. By the third year she should have sufficient background and training to handle other types of disorders. As a result of this type of program the total school system would become more speech conscious, and the speech improvement and correction program would be highly integrated. Although ultimately this program will probably be as expensive as hiring a few well-trained speech correctionists, it will be more easily absorbed by the school system and more people will be directly affected and thus become enthusiastic.

For those of you who are directly confronted with this problem, I would suggest that you study the state laws regarding state aid for special education and rehabilitation programs carried on by the schools. State aid is not offered in some states, whereas in others the total salaries and cost of equipment is carried entirely by the state.

Let me again warn you against all dangers resulting from antagonizing individuals against a corrective program. First, be sure that each child you call a defective speaker has an obvious speech handicap. Second, don't blame the parents or other teachers for causing the child's disorder. Third, don't condemn the school system for not making allowance for the defective child in its program. An enemy at the outset will seldom be enthusiastic at the end.

BOOK REVIEWS

AN INQUIRY INTO THE GENESIS OF POOR READING. By C. C. Bennett. New York: Teachers College Publication, Columbia University (No. 755), pp. 139, 1938. $1.60.

Retarded readers in the second and third grades were compared with their normal classmates and the following variables were best supported: (1) there is some possibility that the position of eldest child in the family is propitious to good reading, (2) children with a history of speech defect seem liable to failures in reading and perhaps a speech therapist should not dismiss as cured a case until all factors conducive to dyxlexia have been removed, (3) teachers tend to regard poor readers as lacking in persistence and in the capacity for sustained and concentrated attention, (4) an inclination to pursue an inactive and solitary life rather than a physically vigorous and gregarious one may be more common among the poor readers and this stands contrary to the common opinion, (5) poor readers tend to regard the school situation as rather unpleasant and difficult to face, and (6) poor readers tend to regard themselves as somewhat inadequately fortified in facing life's difficult situations and tend toward crying spells, fears, indecision, headaches, loneliness, and stuttering.

C. H. V.

AN EVALUATION OF VISUAL FACTORS IN READING. By H. A. Imus, J. W. M. Rothney and R. M. Bear (Dartmouth Medical School). Hanover, New Hampshire: Dartmouth College Publications, pp. 144, 1938.

Exact measurements from Dartmouth Eye Institute, together with statistical treatment by Rothney (Harvard). Required reading for all in the field. It would appear that group methods and group devices may well be used in teaching normal students to read, but it must be admitted that speeded reading does not at all guarantee better comprehension or better academic grades. Insists dyslexia remains a clinical and an individual problem.

C. H. V.

BETTER PRIMARY READING. By C. R. Stone. St. Louis: Webster Pub. Co., pp. 536, 1936.

The aim of this book is to prevent reading disabilities by applying the findings of research. The book is indexed and has a bibliography of 136 items. After a discussion of *Better Reading*, Stone outlines *A Flexible Course of Study*, gives attention to *A Graded Vocabulary for Primary Reading*, and then takes up the *Fundamental Habits* including eye movements. Other chapters include: *Need of Better Balance in Beginning Methods, Level One, Levels Two and Four, Levels Five to Eight, Word Recognition,* and *Activities.* Chapter XI is devoted to the *Prevention, Diagnosis,* and *Instruction* of *Problem Cases in Reading.* Stone has written seven previous books in this field.

C. H. V.

HYPOTHYROIDISM AS AN ETIOLOGY OF CLEFT PALATE

JANET BROWN
(Submitted through Dr. Robert West, Director of Speech Clinic, University of Wisconsin)

Introduction

The problem of the etiology of congenital cleft palate and harelip has received less attention than the operative procedures and post-operative speech correction techniques for the defect.

It is believed that the defect is not due to accident, prenatal impression or malnutrition nor to the degenerative effects of modern civilization. (1) We know that cleft palate is a normal condition at a certain stage of fetal life and that the persistence after birth is the result of arrested development in which two early embryonic halves are prevented in some way from fusing as they normally should; it is by defect of union on the part of the maxillary processes that cleft palate is produced.

This study presents certain facts bearing on the relation between the congenital cleft palate and possible deficiency of the thyroid gland. The results are by no means conclusive, but the author hopes to evoke comment and further interest in the problem.

Observations have been made of skeletal retardation and failure of fusion in hypothyroid cases. The reports have largely concerned animals, but Zondek cites the case of a child of seven with congenital myxoedema who exhibited marked delay in the osseous union.(23) Kennedy observes that roentgenographic examination of the skeleton of the thyroid deficient infant is an aid in diagnosis, and that the cranial sutures fail to close in such a case.(8)

Zondek further states that "so soon as the development of the osseous system has been completed, thyroid deficiency does not seem to give rise to any significant changes, but in the growing bones the signs of deficiency are very pronounced."(23)

Studies of thyroidectomized animals support Zondek's statement. He himself states that a thyroidectomized dog showed a narrow and valuted palate.(23) The growth of skeleton ceases, bones remain short and fragile, calcification is incomplete and fusion arrested. In thyroidectomized rats, a marked sign was the fore-shortened, infantile head.

Perhaps the most extensive studies of skeletal growth in thyroidectomized animals were made by Todd, Wharton and Todd.(17) They compared the skulls of thyroidectomized sheep with those of control animals. One of the experimental animals showed defective growth in the maxilla resulting in an upcurling of its palatal surface

in front; another presented a defect in the vertical growth of the frontal process of the maxilla. In the skull of a third, a fourteen months old animal, the maxillary sutures had not commenced to fuse; and a fourth sheep of twenty-four months showed the same defect. A fifth at twelve months showed maxillary development of three months, and a sixth at thirty-two months showed the same level of maxillary and premaxillary development.

According to Todd, Wharton and Todd, it is in the premaxilla and the anterior part of the maxilla that defective jaw development chiefly shows itself. These regions undergo obvious and permanent defects of growth so that they remain throughout life small and ill-formed with the result that the anterior bony palate is upcurved. The suture of the palate of sheep is normal at two years. Thyroidectomized sheep show no progress in suture-closure beyond that typical of four months.

They concluded that maxillary growth is inhibited after thyroidectomy, having observed that in the skull the most marked developmental disturbance is in the lack of union of the maxilla and the pre-maxilla. In their article they mention Dr. Zuck's study on guinea pigs, in which Zuck pointed out the relationship of hypothyroidism to lapsed union.

This short summary represents the pertinent literature discovered thus far by the author; further research is intended in the hope of finding additional material.

Original Findings

It is well known that there are "thyroid areas"—regions in which thyroid dysfunctions are particularly prevalent. These areas are shown on the accompanying map which designates the number of goiter cases per thousand population, and also the iodin content of the water in the outlined regions. The thyroid regions follow closely the areas of glaciation in the United States, i. e., there is a positive correlation between glaciation and a high incidence of thyroid disturbances.

On the basis of this fact, a preliminary study was made of the largely glaciated state of Wisconsin and the largely non-glaciated state of New Jersey. *It seemed logical to believe that if congenital cleft palate were a result of a thyroid dysfunction, it would be more prevalent in a glaciated state.*

The two states are not ideal geographically for such a study, since they cannot be exactly contrasted on the basis of glaciation alone. One of them is an inland state, and the other coastal, and one partially mountainous while the other is flat, but they are the only two states requiring the reporting of congenital defects on the birth certificate.

The laws themselves are comparable. Wisconsin's law provides for enforcement by the local registrar and states that: "Within twenty-four hours after the birth of any child with a deformity or

physical defect, the attending physician or midwife, or if there is no physician or midwife in attendance, the parent or guardian, shall in addition to and separate from the notice thereof required in the birth certificate directly notify the state board of health of such deformity or defect, and shall explain as fully as possible the exact nature thereof." The penalty should be, "a fine of not less than twenty dollars or more than two hundred dollars, or imprisonment in the county jail for not less than thirty or more than sixty days—or both."(16)

The birth reporting law for New Jersey requires that: "Within thirty days after the date of the birth of any child born in this state with visible congenital deformities, the physician, midwife or person acting as midwife, who shall be in attendance upon said birth, shall prepare and file with the commission for the care and treatment of crippled children—a statement setting forth such visible congenital deformities."(9) The penalty for failure to report is a fine of fifty dollars.

With laws and penalties that strict, it seems justifiable to assume that the statistics of both states are reliable, and also comparable in that respect.

The maps of the two states are, the author believes, self-explanatory and will show that in proportion to the total areas of the states, the driftless or non-glaciated area of Wisconsin is equal in size to the glaciated area of New Jersey.

The following figures were obtained from the Crippled Children Commission[1] and the report of the State Health Department in Wisconsin. The term "cleft palate" includes harelip, cleft palate, and harelip and cleft palate deformities. The three types are listed separately in the reports of both states, but are combined here and considered as degrees of the same basic defect.

WISCONSIN

Year	Live Births	Cleft Palate	No. per Thousand
'32–'33	103,776	113	10.9
'34–'35	104,166	110	10.7
'36–'37	106,332	120	11.3

NEW JERSEY

Year	Live Births	Cleft Palate	No. per Thousand
'32–'33	117,287	56	4.8
'34–'35	109,900	41	3.7
'36–'37	109,342	65	5.9

These tables show that there are more than twice as many cleft palate births per thousand in Wisconsin as in New Jersey.

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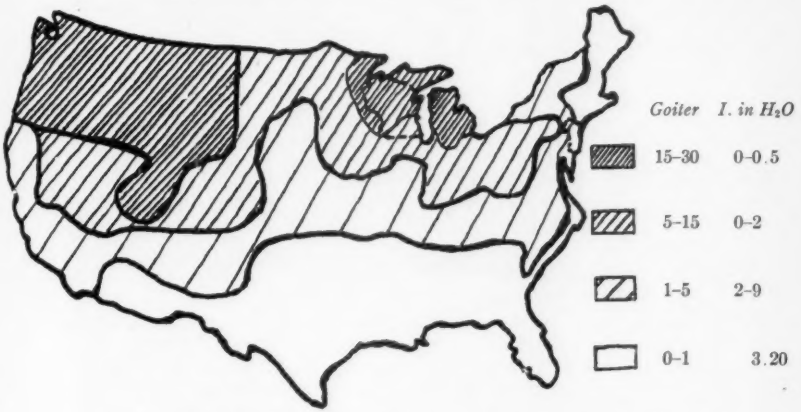
[1]This material was obtained through the co-operation of Miss Gertrude Miller, secretary of the Crippled Children Commission of New Jersey.

BOUNDARIES OF WISCONSIN AND NEW JERSEY

Discussion

As the figures stand they appear to support the contention that there should be more cleft palate births in a glaciated area if the deformity is the result of a thyroid deficiency. There are, however, qualifying factors to be considered.

First, determining by observation of the maps, the rough proportion of the several counties in New Jersey that are glaciated, and the counties in Wisconsin that are in the driftless area, and then finding their total populations, it was found that 65.8% of the people of New Jersey live in the glaciated area, while only 16.6% of the population of Wisconsin resides in the driftless area.



Comparison of iodin in water supplies and distribution of goiter: iodin in parts per billion of representative rivers; goiter per thousand from Table xxxiii, Defects Found in Drafted Men, War Department, 1920, and manuscript; curves smoothed.

Map from McClendon, J. F., *Journal of American Medical Association*, March 3, 1923, p. 600.

The next step will necessarily be to attempt to discover from a study of the birth certificates, just how many of the cleft palate births were in those areas. Until those statistics are obtained, it must be a matter of conjecture as to whether the higher rate of cleft palate births can be attributed to the factor of glaciation. On the other hand, the three counties in New Jersey most densely populated, namely Essex, Hudson and Bergen, are shown on the goiter map to be in the region of lowest incidence for that defect.

The second factor to be taken into account is the proximity to sea water. J. F. McClendon (11) states that iodin is distributed from sea spray, and New Jersey being a coastal state has that advan-

tage. In addition, its position is advantageous because of the greater opportunity for consumption of sea food.

McClendon further reports that the burning of coal is a factor in iodin distribution, (11) and we find that the coastal section of New Jersey's glaciated area is predominantly an industrial region.

These facts make it impossible to draw conclusions from the comparison of the cleft palate births, but it seems unlikely that the sea spray and sea food advantages could account for the entire difference in number of cleft palate births.

Aside from this study it is interesting to note the statement that Beatty (1) makes about the alternation of generations born with cleft palates. He observes that the hereditary tendencies cannot be ignored. One generation may escape though the defect may appear in both the preceding and following generations. This may be significant in view of the fact that a hypothyroid child may be born to a hyperthyroid mother, and a hyperthyroid child to a hypothyroid mother, establishing an alternation of generations in this respect. This comparison too is presented as a suggestion for future study.

Conclusions

It has been our purpose to suggest that congenital cleft palate may be the result of a thyroid deficiency. As a first step in testing this hypothesis we have noted the high incidence of the palatal deformity in the glaciated or thyroid area, and the low incidence in the non-glaciated area.

It is our hope that additional figures on cleft palate births will be made available by state laws requiring the reporting of congenital deformities, and also that further studies will be made in the effort to find the cause of the defect, the importance of which is stated by Dr. Robert West in this way: "Cleft palate causes a greater alteration of speech sounds than does any other malformation. In severe cases the speech is practically unintelligible."(19)

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ANNOUNCEMENT

The Congress of the Internationale Gesellschaft für Logopadie und Phoniatrie will meet in the summer of 1940 in Amsterdam, Holland. President Herman Gutzmann will preside.

BOOK REVIEWS

PERSONAL-SOCIAL ADJUSTMENT TEST. By Elwood Murray and L. W. Miller (University of Denver).

The present test is a speech test of speech and measures egocentric introversion, egocentric extraversion, mentally objective introversion and mentally objective extroversion in relation to speech situations. Few were better equipped to devise such a test than Murray.

C. H. V.

THE ADOLESCENT PERIOD, A GRAPHIC AND PICTORIAL ATLAS. By F. K. Shuttleworth (Yale University). Washington: Society for Research in Child Development of the National Research Council, Vol. 3, No. 3, 1938.

246 pages with 458 figures completely cross indexed. For those of us who work principally with high school and college adolescents. Each figure is based on the best research available concerning that problem and each one is copied directly from the primary source. The selections are made by an expert in the adolescent field. The central tendencies and the individual variations both are amply illustrated. Reference information about the growth of the palate, teeth, vital capacity, reading ability, etc.

C. H. V.

AN INTRODUCTION TO THE PHONETIC ALPHABET. By S. T. Barrows. Boston: Expression Co., pp. 55, 1938. $1.00.

Phonetics is one of the bases of speech correction and this little manual removes all that is sometimes considered complicated and cumbersome in using the phonetic alphabet. The book is planned for the embryo speech pathologist and takes very slow steps, giving mastery to each before the next is taken.

C. H. V.

LIVING SPEECH. By G. L. Borchers. New York: Harcourt, Brace and Co., pp. 289, 1938. $1.32.

This is the University of Wisconsin secondary school speech text, but is also planned for use as a part of English courses. Borchers believes that the fundamental processes of speech are: *action, voice, language* and *pronunciation,* which is similar to Woolbert's list except *pronunciation* appears where Woolbert used *thought.* These fundamentals are taught through developing skill in *pantomime, interviews, club meetings, conversation, telephoning, introductions, parliamentary practice, classroom recitations,* and *talks.* It is believed that the student will discover his own speech problems and their solution by analyzing the behavior of himself and his classmates in these speaking situations.

C. H. V.

CHINESE DIFFICULTIES WITH ENGLISH PRONUNCIATION

C. K. THOMAS,
Cornell University

The Chinese students in the United States represent a wide variety of linguistic types. In the first place, their Chinese linguistic backgrounds vary; regional types of speech differ more sharply from each other in China than in the United States. Thus the basic phonetic habits of the individual Chinese student differs from those of his fellows, and may be presumed to complicate the problem of teaching him to speak English. Actually, however, regional differences are of little practical importance. The same sorts of difficulties are evident in the English speech of Chinese students with quite different regional backgrounds.

More important are native linguistic ability and the kind of English training the individual has received in China, since almost all the students who come to this country have had several years of previous English study. At the one extreme we find the linguistically competent Chinese whose speech is indistinguishable from that of the native American; at the other, the recent arrival whose training in English has been so sketchy that he can barely get along without an interpreter. Most of the Chinese students who have worked in the Speech Clinic at Cornell University during the past ten years have had sufficient command of English to make themselves understood without great difficulty. All have had difficulties with English phonemes—difficulties which, in retrospect, seem characteristic, and which form the subject matter of this article.

Though stopped consonants offer less difficulty than others, certain characteristic substitutions may be noted. Initially and medially, [pbtdkg] are usually normal. There is little noticeable dentalizing, no noticeable palatizing, and little confusion between the aspirated and unaspirated forms of [ptk]. Consecutive stops are not, however, usually linked, as in native English, but are separated by an aspirate or a vowel. Thus, *bookcase* may be heard as [bʊkhkes] or [bʊkəkes]. At the end of words the Chinese habitually substitutes the voiceless [ptk] for the voiced [bdg], and occasionally the glottic stop for any final stopped consonant.

In Chinese, nasal consonants commonly come at the end of the word, and there is a wealth of nasal vowels. It is therefore confusing to find nasal consonants in various positions within the English word, and an absence of nasal vowels. At the end of words the Chinese frequently substitutes a nasal vowel for any English nasal consonant.

In other positions, [m] is usually normal, but [n] and [ŋ] readily exchange places with each other, and evidently represent but one phoneme to the Chinese. Thus, [paŋd] and [sɪnə] are characteristic pronunciations for *pond* and *singer*. An additional confusion between [n] and [ŋ] results from the Chinese tendency to produce a nasal consonant by simultaneous elevation of the tip and the back of the tongue. This minimizes the difference between [n] and [ŋ], and increases the likelihood that a nasal vowel will be substituted. This double action also underlies the well known confusion between [l] and [r], as we shall see later.

Less frequently, medial [ŋ] acquires a parasitic [g], and *singer* becomes [sɪŋgə]. This is probably not a phonetic tendency, but an effect of the awkward representation, in English, of both [ŋ] and [ŋg] by means of the letters "ng." Before medial stops, [mnŋ] may themselves be parasitic. *Puppy* may become [pœmpɪ], and *bigger* [bɪŋgə]. Parasitic nasals are less frequent before other consonants, though I have heard [pənθɛtɪk] for *pathetic*. In some circumstances, as we shall see later, [n] may be substituted for [l].

Fricatives are also highly variable. When final, [vðz] are usually unvoiced to [fθs], similarly to the unvoicing of final stops. [ʍ] may become [f] or [v]; *white* becomes [faɪt]; *whistle* becomes [vɪsl]. Substitution of [w] for [ʍ] is, however, less common. [f] is relatively stable, but may become [ʍ], as in [ʍæt] for *fat;* less frequently it may be bilabial, as in Japanese, instead of labiodental. [v] is usually stable, but may become [w]; I have heard [ʃɪwət] for *shivered.*

The dental and alveolar fricatives are highly variable. [θ] is overtense at best. More often it becomes [s], occasionally [t]. Thus, [sæŋk] may be heard for *thank*, and [tɪn] for *thin*. Unlike [θ], the voiced [ð] occurs somewhat more frequently as a stop than as a fricative. Most commonly it becomes [d], as in [dɪs] for *this;* less frequently it becomes [z], as in [wɪz] for *with*. In the medial position, [ð] sometimes is unvoiced, as in [wɪθə] for *wither*. The usual Chinese [s] is retracted, in comparison with English, and acoustically intermediate between English [s] and [ʃ]. When this Chinese [s] is used in English words, it sounds muffled; sometimes it passes completely over to [ʃ], and *singer* becomes [ʃɪŋə]. Chinese [z] is also retracted, but less noticeably so, probably because of the masking effect of voice. The Chinese have little trouble with English [ʃ], though some form of [s] may occasionally be substituted. I have heard [si] for *she*. The voiced [ʒ], on the other hand, is very troublesome. Most frequently it becomes [ʃ], as in [plɛʃə] for *pleasure*. Less common substitutions are [z] and [s], as in [trɛzə] for *treasure* and [vɪsn] for *vision;* spelling pronunciation may, of course, play a part here. Chinese [h] is a more vigorous sound than its English counterpart; often it approaches the tenser [x], and becomes roughly the equivalent of German or Scotch "ch" or of Spanish "j."

The Chinese treatment of the affricates [tʃ] and [dʒ] shows a certain parallelism to the treatment of the simple fricatives [ʃ] and

[ʒ]. [tʃ] is usually simplified to [ʃ], as in [kɪʃɪŋ] for *kitchen*. Attempts to restore the [t] are quite as likely to result in [ʃt] as in [tʃ]. [dʒ] follows the characteristic development of being unvoiced to [tʃ], as in [etʃ] for *edge*, and may then go on to vary in any of the ways already noted for variants of [tʃ] and [ʃ].

The semivowels [w] and [j] show a certain parallelism in their variations, especially in those phonetic contexts in which they are sometimes lost altogether. Thus, [w] may be lost before relatively high back vowels, and [j] before high front vowels, as illustrated by the following characteristic pronunciations: *won't* [ont], *woods* [ʊts], *woo* [u], *yeast* [ist]. Both [w] and [j] are weak sounds; to the Chinese their similarity to [u] and [i] evidently outweighs the differences. He does not clearly hear the difference between [w] and [u], nor between [j] and [i]; at best, [wu] and [ji] are merely more emphatic ways of beginning [u] and [i]. Consequently he remains uncertain about [w] and [j] except when the following vowel is clearly different. Even in such cases some uncertainty remains, and [j] may be dropped or added as a matter of emphasis rather than as an independent phoneme. Thus we may hear [uθ] for *youth*, [jitʃ] or [jiʃ] for *each*, [jɪŋstənt] for *instant*. To complete the parallelism, there should be some instances of intrusive [w]; to date, however, I have heard none.

In English, [r] and [l] have the effect of semivowels when nonsyllabic, and of vowels when syllabic as in *batter* and *battle*. The differences between these semivowels and vowels are, however, slight; for our present purpose they may be disregarded. In popular tradition, the Chinese pronounces [r] when we expect [l], and [l] when we expect [r]. This reversal, when combined with a monotonous high pitch is usually considered adequate to characterize the "broken English" of the Chinese in dramatic representations. It is not, however, adequate for the teacher who wishes to help the Chinese student learn to speak English effectively. Though [l] and [r] may be reversed, the problem is usually more complicated. We have already noted the double articulation which characterizes the Chinese attempt at [n] and [ŋ]. A similar double articulation causes some of the confusion between [l] and [r]. Many Americans articulate [r] by means of a raising of the middle part of the tongue toward the hard palate. In this position the arch of the tongue is further back in the mouth than for [j], but not so far back as for [w] or the uvular [R]. A little practice will enable most Americans to combine this central [r] with the activity of the tip of the tongue which produces [l]. The combined position is, however, relatively unstable; if the tip drops slightly, the sound is more like [r]; if the central part drops slightly, the sound is more like [l]. It is this wavering of the tongue position which produces much of the Chinese difficulty with English [r] and [l], since the Chinese tends to form both English phonemes with the same double articulation. Finally and before consonants, the Chinese quite sensibly omits [r]. But no standard English dialect permits this treatment of [l]. In his efforts to retain some trace of [l] in the relatively weak final and preconsonantal position, the Chinese may

succeed in avoiding any confusion with [r], but is likely to produce instead an unrounded [u] or a variety of [ə]. Before [dr], and possibly in other contexts, [l] may be changed to [n], as in [tʂɪndrən] for *children* and [mɪndrəd] for *Mildred*. A few Chinese have attempted to solve the [l-r] problem by trilling [r], but the trill is not nearly as common among the Chinese as among the Siamese, who, along with the Japanese, also have [l-r] difficulties.

English vowel sounds are so numerous and variable as to cause great difficulty for any foreigner. English has about twice as many vowels as Italian or Spanish, including some varieties which, though non-phonemic, are essential to oral style. The pronunciation of such words as *ask* and *orange* varies widely in good usage, and a change of stress changes the vowel pattern of whole words. English spelling is bewildering, particularly in the representation of such sounds as [ə] and [ɜ]. It is therefore no wonder if foreign attempts at English vowels, including Chinese attempts, result in few chance hits and many misses. For most vowels, the Chinese range of error is likely to be limited to nearby vowels. That is, in attempting [ɪ], he may produce [i] or [e]; in attempting [æ], he may produce [ɛ] or [a]; in attempting [ə] and [ɜ], the result will depend on the sort of analogy which the spelling suggests at the moment. In all this variation, his errors are not essentially different from those of other foreigners. In contrast with other foreigners, however, a few facts are noteworthy. The Chinese has less difficulty with English [a], [a :], and [u] than with other English vowels, and his most common substitute for [ʌ] is characteristic. Chinese has no [ʌ], but it does have an [œ] which is roughly the equivalent of the vowel of French *neuf*, and this [œ] serves as the commonest Chinese substitute for [ʌ]. Other substitutions, such as [a], [ɔ], [ʊ], and [u], though less common, indicate that the Chinese grasps English [ʌ] less readily than other vowels.

Although Chinese has approximate equivalents for all the major English diphthongs except [ɔɪ], Chinese speakers regularly minimize, and often eliminate, the second elements of English diphthongs. As with the vowels, the first elements usually waver in the direction of neighboring sounds, but there are no startling changes in the first elements. Thus [eɪ] and [oʊ] commonly occur as [e :] and [o :], as they do in some contexts in American speech. [aɪ] may occur as [aɪ], [aə], [a :], [aə], or [a :], as well as in the normal form. [aʊ] is almost never normal; its commonest form is [a :], as in [da : n ta : n] for *down town*. The first element may shift toward [ɒ] and [ɔ], but never toward [a] and [æ], as in American speech. The treatment of [aʊ] is perhaps the most noticeably characteristic feature of Chinese attempts at English diphthongs. [ɔɪ] is usually pronounced without the second element, as in [ɔ : l] for *oil*, a pronunciation which the Chinese shares with many American Southerners. The first element wavers slightly, but never toward the centralized [ɜɪ] of New York City.

In comparison with the difficulties of other foreigners, the most characteristic Chinese pronunciations of English are those of [s], [n],

[ŋ], [l], [r], [j], [w], [ʌ], and [aʊ]. In any remedial program, these phonemes should have a prominent place. Though other phonemes may actually cause more difficulty, they will not so definitely identify the foreign pronunciation as Chinese pronunciation.

BIBLIOGRAPHICAL NOTE

Except for a short section (pp. 144–6) in *The Teacher's Book of Phonetics* by Sarah T. Barrows and Anna D. Cordts (Boston, Ginn and Co., 1926), I know of no systematic account of Chinese difficulties with English. For the background material of Chinese phonetics, however, there exist many volumes, of varying degrees of accuracy. For the speech correctionist, the following volumes will probably be most useful:

1. **Bernhard Karlgren.** *Etudes sur la Phonologie Chinoise*, a series of four monographs published by the *Archives d'Etudes Orientales*. Upsala, 1915–1926. These studies, which deal with 26 of the dialects, are the most complete account of Chinese pronunciation with which I am familiar.
2. **Bernhard Karlgren.** *A Mandarin Phonetic Reader in the Pekinese Dialect*. Upsala, 1917.
3. **Daniel Jones** and **Kwing Tong Woo.** *A Cantonese Phonetic Reader*. London, 1912.
4. **Yuen Ren Chao.** *A Phonograph Course in the Chinese National Language*. Shanghai, 1925.

Jones and Woo and Chao use the alphabet of the International Phonetic Association; Karlgren, a more complicated alphabet of his own.

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† WILHELM BERGER †

MAX NADOLECZNY

Am 2. Juli 1938 ist uns Wilhelm Berger genommen worden. Er starb den Fliegertod, den Tod fürs Vaterland, für das er so oft sein Leben eingesetzt, für das er gekämpft, gelitten, gelebt hat. Erschüttert von der Tragik unserer Zeit standen Lehrer, Schüler, Freunde an der Seite einer jungen Witwe und der Kinder in tiefer Trauer an seiner Bahre. Ein allzu kurzes Leben hat einen jähen Abschluss gefunden. Ein Mann ist uns genommen worden, welcher der Wissenschaft und damit auch dem Ansehen seines Vaterlandes in normalen Zeiten noch wertvolle Dienste hätte leisten können.

Wilhelm Berger wurde am 11. Oktober 1895 zu Herford i/Westfalen geboren und hat das Schiller-Gymnasium zu Münster i/W. besucht, wo er im März 1914 das Reigezeugnis erhielt. Im April ist er in den Heeresdienst getreten, und im Sommer ins Feld gezogen. Dreimal ist er im Weltkrieg schwer verwundet worden.

Erst im Sommersemester 1919 konnte er sein medizinisches Studium aufnehmen, 1922 folgte das medizinische Staatsexamen, 1923 die Promotion in Hamburg. 1923 war er Assistent am Pathol-

ogischen Institut, 1924–25 an der Hals-Nasen-Ohrenklinik in Hamburg. 1925/26 machte er als Schiffsarzt Reisen nach Südamerika und Niederländisch-Indien. Von Oktober 1926 bis 1934 war er Assistent an der Abteilung für Sprach-und Stimmkranke der Poliklinik in München und an den oto-rhino-laryngologischen Kliniken in Würzburg und Münster i/W., wo er sich im Juli 1931 habilitierte und zwar mit einer phonetischen Arbeit "über normale und pathologische Gleitlaute." (Glide) Im November 1934 ist er als o. Professor und Direktor der Hals-Nasen-Ohrenklinik an die Universität Königsberg berufen worden. Dort war er Dekan der Medizinischen Fakultät seit 1935.

Seine besonderen Leistungen liegen auf dem Gebiet der Sprach- und Stimmheilkunde. Hier sah er Neuland, auf dem es zu forschen gab, Fragestellungen, die durchaus nicht nur theoretische, sondern mit praktischem Erfolg verwertbare Lösungen versprachen. Schon die erste kasuistische Arbeit über doppelte Stimmlippen"(Zeitschr. Hals-usw. Heilk. 1928) zeigt seine Vertrautheit mit phonetischen Untersuchungsverfahren. Die Bedeutung der Mitteilung über "Kehlkopftrauma mit Abriss beider Stimmlippen" (Passows Beiträge 1930) besteht nicht sowohl in der klinischen Beschreibung des Falles, als vielmehr darin, dass Berger denselben benutzt hat, um einen Beitrag zur Vokaltheorie zu liefern: Die Entstehung—allerdings heiserer—Vokale kann auch auf aperiodisches Anblasen des Ansatzrohrs erfolgen. In den Vorträgen "Experimentelles über die Vokaltheorie" 1932 und "über Vokaltheorien" 1936 (Arch. für Sprach-u. Stimmheilkunde 1937) hat er immer wieder zu dieser wichtigen Frage der Phonetik auf Grund eigener Forschungen Stellung genommen. Auch in der Arbeit "über subglottische Druckmessungen an Kanülenträgern" (Z. Laryngol. 1934) wurden pathologische Zustände benutzt, um physiologische Fragen: Einfluss des Atemvolumens auf den subglottischen Druck usw. anzugehen mit dem Ergebnis, dass aus den gewonnenen Werten auf mechanische Vorgänge im Kehlkopf nicht geschlossen werden darf, wie das von anderer Seite versucht worden war. Durch Klanganalysen gelang es Berger, die Gutzmannsche Druckprobe (Z. Laryng. 1934) zu einem genauen diagnostischen Untersuchungsverfahren auszuarbeiten und ihre bisher nur empirische Verwendung zu begründen. "Klangaufnahmen bei Diplophonie" (Z. Laryng. 1933) und die "Beiträge zur Sprachklanganalyse" (Z. Biol. 1934) zeigen, wie er, weit mehr als der Durchschnitt selbst der Fachphonetiker, die schwierigen mathematischen Fragen beherrscht hat und daher in der Lage war, an der damals—und z. T. auch heute noch—ziemlich verworrenen Lehre von der Analyse der Klangkurven klärend mitzuarbeiten. In diesen Veröffentlichungen sehe ich nur grundlegende Vorarbeiten zu einer exakten Pathologie, der Phonation, wozu die Klangaufnahmen bei Diplophonie den Anfang bilden (Schwebungs- und Stosskurven und deren Analyse), während die Beiträge zur Analyse pathologischer Stimmklänge (Arch. f. Sprach-u. Stimmheilkunde 1937) die Fortsetzung bilden.

(*Continued on page 266*)

A LATERALITY STUDY OF NON-STUTTERERS AND STUTTERERS[1]

CLARENCE R. VAN DUSEN, D.Sc.
Michigan State College,
East Lansing, Michigan

The theory that there is a lack of dominant lead in the stuttering person has occasioned much testing of handedness. The question has arisen as to whether any significant difference in handedness exists between non-stutterers and stutterers (4,5). In connection with a study, of the relation of the relative size of the two hands to speech, the author (4, 5) conducted an investigation of the handedness of eighty college males.[2] Laterality tests were administered to forty right-handed non-stutterers and forty right-handed stutterers to discover whether any significant differences in handedness exist between the two groups, and to test further the brain dominance theory regarding stuttering.

Apparatus and Experimental Procedure

A questionnaire study of hand usage and tests of strength, speed, accuracy, and laterality lead were given. Hand clasp and eye fixation were studied also. The questionnaire of hand usage, and the procedure used in filling it in and scoring it, was similar to that of Koch (3) and Van Riper (6). Sample questions from the usage questionnaire follow:

1. When playing the ukelele, with which hand do you prefer to strike the strings?.....R L E
2. When firing a gun, with which hand do you manipulate the trigger?.....R L E
3. When rubbing clothes which you are washing, which hand does most of the work?.....R L E

Dextrality quotients[3] were determined by use of the formula:

$$D.Q = \frac{R + \frac{B}{2}}{N}$$

[1]The author wishes to express appreciation to Professor John H. Muyskens, Professor Clarence L. Meader and Mr. Robert E. Card for assistance so graciously offered in this investigation and to Professor William D. Baten for careful guidance in statistical procedures.

[2]Characteristics of cases are presented in other writings by the author (4, 5).

[3]The dextrality quotient has been described by Johnson, W. and Duke, D. (2) and has been defined as "The total percentage of total achievement involved in any test of handedness which can be credited to the right hand."

where R and B represent the number of operations performed by the right hand and by both hands (neither hand predominating), respectively, and N represents the total number of operations.

The Collins' Dynamometer was used in testing for strength. Apparatus devised by the author was used in testing the relative speed and accuracy of the two hands.[4]

In testing for laterality lead, a critical angle board was employed at a position perpendicular to the chest surface of the subject. The procedure used was similar to that employed by Van Riper (6). To make findings obtained in this part of the study comparable with findings from the questionnaire study of hand usage, the dextrality quotient was used. In the formula, R became the number of right hand leads, B became the number of conflicts between the two hands, and N the number of tests administered. Percentages obtained have been called "dominance quotients."

Eye fixation was tested by having the subject look at the experimenter's nose through a hole in a card.

Findings

Absolute and percentage frequencies of cases revealing dextrality, sinistrality, and ambidextrality are shown in Table I. Dextrality quotients[5] of non-stuttering subjects were 75.0 per cent or higher in 97.5 per cent of the cases. The dextrality quotients of stuttering males were 75.0 per cent or higher in 100 per cent of the cases. Dominance quotients of non-stuttering subjects were 75.0 per cent or higher in 92.5 per cent of the cases. Dominance quotients of stuttering subjects were 75.0 per cent or higher in 82.1 per cent of the cases. In this test, 17.9 per cent of the stuttering group were found to be ambidextrous, while only 7.5 per cent of the non-stuttering group were found to be ambidextrous.

A larger per cent of non-stutterers than stutterers placed the right thumb uppermost in the hand clasp test. Only one of the non-stutterers was found to have a stronger left hand while in the stuttering group there were six subjects with a stronger left hand. In the test of speed, left hands were found to be more agile than right hands. Right hands were found to be more agile than left hands in a greater per cent of the stuttering subjects than of the non-stuttering subjects. Findings in the accuracy test were quite similar for both groups. There was a slightly greater per cent of cases in the non-stuttering group having more accurate right hands than left hands than there

[4]Apparatus and experimental procedures used in the testing program have been fully described in a thesis by the author (4).

[5]In the questionnaire study of hand usage, subjects were considered to be right-handed if the dextrality quotient was 75.0 per cent or higher, and left-handed if the dextrality quotient was 25.0 per cent or lower; otherwise, the subject was considered ambidextrous. This same procedure was used with dominance quotients.

was of stutterers. A slightly greater per cent of stutterers was found to be right-eyed, than was non-stutterers.

As far as findings shown in Table I are concerned[6] if it were assumed that the stutterer, as opposed to the non-stutterer, tends toward disintegrated handedness and eyedness, the assumption would prove false in findings for usage, eye fixation, and speed. There is a greater tendency toward sinistrality and ambidextrality in the hand clasping test, tests of strength, accuracy and laterality lead in the stuttering group than in the non-stuttering group.

TABLE I

THIS TABLE SHOWS THE ABSOLUTE AND PERCENTAGE FREQUENCIES OF RIGHT-HANDED NON-STUTTERING MALES AND RIGHT-HANDED STUTTERING MALES REVEALING DEXTRALITY, SINISTRALITY, AND AMBIDEXTRALITY IN THE TESTS OF LATERALITY

Test	Number of Cases	Right		Left		Either	
		No.	Per Cent	No.	Per Cent	No.	Per Cent
Right-handed non-stuttering males:							
Statement	39	39	100.00	0	.00	0	.00
Writing	39	39	100.00	0	.00	0	.00
Usage Questionnaire	39	38	97.50	0	.00	1	2.50
Hand Clasp	39	22	56.00	16	41.00	1	3.00
Strength	40	39	97.50	1	2.50	0	.00
Speed	40	14	35.00	24	60.00	2	5.00
Accuracy	39	31	79.50	7	17.90	1	2.60
Brain dominance	40	37	92.50	0	.00	3	7.50
Eye fixation	40	29	72.50	11	27.50	0	.00
Right-handed stuttering males:							
Statement	40	40	100.00	0	.00	0	.00
Writing	40	40	100.00	0	.00	0	.00
Usage Questionnaire	37	37	100.00	0	.00	0	.00
Hand Clasp	39	17	43.60	22	56.40	0	.00
Strength	37	31	83.70	6	16.30	0	.00
Speed	40	17	42.50	20	50.00	3	7.50
Accuracy	40	31	77.50	8	20.00	1	2.50
Brain dominance	39	32	82.10	0	.00	7	17.90
Eye fixation	39	29	74.40	10	25.60	0	.00

Table II shows mean per cents of laterality quotients obtained in a study of the two groups. In comparing these means (see Table III), no significant differences were found in tests of laterality lead. The t-score, 2.2292, indicates that there was a significant difference between non-stutterers and stutterers for usage, the stutterers tending to be more definitely right-handed.

[6]These findings show merely the number of cases tending toward dextrality, sinistrality or ambidextrality. From them no determinations regarding significance of difference between the two groups can be obtained.

A study was made to determine whether or not there was a significant difference between the two groups in strength, speed, and

TABLE II

THIS TABLE SHOWS MEAN PER CENTS WITH STANDARD DEVIATIONS OF DEXTRALITY QUOTIENTS AND DOMINANCE QUOTIENTS OBTAINED IN A STUDY OF RIGHT-HANDED NON-STUTTERING MALES AND RIGHT-HANDED STUTTERING MALES

Test	Number of Cases	Mean Per Cent	Standard Deviation
Non-stutterers:			
Usage Questionnaire..... (Dextrality Quotient)	39	91.4 ± 1.1825	7.2892 ± 0.8361
Brain Dominance..... (Dominance Quotient)	40	92.0 ± 1.6449	10.2726 ± 1.1631
Stutterers:			
Usage Questionnaire..... (Dextrality Quotient)	37	94.6 ± 2.4581	14.7486 ± 1.7381
Brain Dominance..... (Dominance Quotient)	39	86.3 ± 2.7790	17.1306 ± 1.9650

TABLE III

THIS TABLE SHOWS THE FISHER'S T-SCORES (1) OBTAINED IN COMPARING THE MEANS FOR RIGHT-HANDED NON-STUTTERING MALES AND RIGHT-HANDED STUTTERING MALES OF LATERALITY QUOTIENTS. THE T-SCORE INDICATES THAT THERE WAS A SIGNIFICANT DIFFERENCE BETWEEN THE TWO GROUPS IN TESTS OF USAGE. NO SIGNIFICANCE WAS OBTAINED IN TESTS OF LATERALITY LEAD

Quotient	Number of Cases	t
Usage Questionnaire..... (Dextrality Quotient)	74	2.2292
Brain Dominance..... (Dominance Quotient)	77	1.4744

TABLE IV

THIS TABLE SHOWS THE FISHER T-SCORE (1) OBTAINED IN A STUDY TO DETERMINE THE SIGNIFICANCE OF THE DIFFERENCE BETWEEN RIGHT-HANDED NON-STUTTERING MALFS AND RIGHT-HANDED STUTTERING MALES IN TESTS OF STRENGTH, SPEED AND ACCURACY. THE T-SCORES INDICATE THAT THERE WAS A SIGNIFICANT DIFFERENCE BETWEEN THE TWO GROUPS IN TESTS OF STRENGTH. NO SIGNIFICANCE WAS OBTAINED IN TESTS OF SPEED AND ACCURACY

Test	Number of Cases	t
Strength.....	75	2.4966
Speed.....	78	0.8940
Accuracy.....	77	0.1776

accuracy. The difference between the right hands and the left hands of the non-stutterers was compared to the difference between the right hands and the left hands of the stutterers. Fisher's formula (1)

for comparing two means for separate groups was applied. Results obtained are presented in Table IV. The t-scores indicate that there was a significant difference between the two groups in tests of strength. This difference shows the non-stutterers to be more definitely right-handed in regard to strength than the stutterers. No significant difference was obtained for speed or accuracy.

Summary and Conclusions

1. Findings from this battery of tests would indicate that significant differences in laterality between non-stutterers and stutterers are present only in strength and usage.
2. Differences in strength show the non-stutterers to be more definitely right-handed than the stutterers.
3. Differences in usage show the stutterers to be more definitely right-handed than the non-stutterers.
4. There are no significant differences between the two groups in laterality lead, speed, and accuracy.

As far as the tests administered to these two groups of cases are concerned, findings do not support the assumption that there is a lack of dominant lead in the stuttering person.

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LET'S READ. By H. Roberts and H. Rand. New York: Henry Holt & Co., pp. 598, 1937. $1.44.

"Let's read is based on the conviction that few persons begin to read well until they find enjoyment and satisfaction in reading." It does not need to be confined to the seventh and eighth grades. Practice material for silent or oral reading.

C. H. V.

WILHELM BERGER

(*Continued from page 260*)

Dem praktischen Spezialarzt näher liegen die Aufsätze über "Organisches und Funktionelles bei Stimmstörungen" (Z. Laryng. 1929), über den "Wechsel organischer Ausdrucksformen bei funktionellen Stimmstörungen" (ebenda 1932), über echten und vorgetäuschten Lombardschen Versuch" (Monatschr. f. Ohrenheilk. 1932) sowie "über vorgetäuschtes Ablesen" (ebenda 1931). In diesen Arbeiten hat Berger psychiatrische Betrachtungsweisen in die Lehre der Stimmpathologie eingeführt. Dabei ist er zu neuen Gesichtspunkten gekommen, indem er den pathogenetischen Krankheitselementen die pathoplastischen gegenüberstellte. Die beiden letzterwähnten Veröffentlichungen enthalten sorgfältig durchdachte neue Verfahren zur Entlarvung von Simulanten und beweisen, dass phonetische Kenntnisse sowie praktische Erfahrungen beim Ableseuntericht Schwerhöriger sehr wohl zu Fortschritten auf gerichtlich-medizinischem Gebiet führen können.

Wenn es erlaubt ist, von einem wissenschaftlichen Vermächtnis bei einem noch so jungen Gelehrten zu reden, so war es dieses: Die Eingliederung der Sprach- und Stimmheilkunde in die praktische Laryngologie und die Erweiterung halsärztlichen Denkens in neurologisch-psychiatrischer Richtung.

Im Jahre 1936 wurde Wilhelm Berger I. Vorsitzender der deutschen Gesellschaft für Sprach- und Stimmheilkunde, im Jahre 1938 Präsident der internationalen Gesellschaft für Logopaedie und Phoniatrie. Vom Jahre 1937 an hat er das Archiv für Sprach- und Stimmheilkunde herausgegeben. Er sollte der deutschen Sprach- und Stimmheilkunde, der *Gutzmannschen* Schule, ein Führer und Förderer für die Zukunft sein. Er war um seiner Liebe zur Wissenschaft und um seines liebenswerten, bescheidenen Wesens willen der geeignete Mann dazu. Fast schüchtern erschien er bisweilen und niemand hätte hinter ihm den Flieger gesucht, der neben der wissenschaftlichen Tätigkeit immer und immer wieder—fast möchte man sagen die Gefahr suchend—im Dienste des Vaterlandes tätig war. Nur wer ihn einmal beim Segelsport im Kampf mit den Elementen des Sturmes leuchtenden Auges der Gefahr trotzen sah, wusste von diesem zweiten Professor Berger, der ein so ganz anderer gewesen ist. Er hatte, wie einer seiner Freunde gesagt hat, die seltene Gabe "Entscheidendes weniger in Worten als mit den Augen auszudrücken." "Wir wollen ihn nie vergessen," hat sein Lehrer Professor Marx-Würzburg, der Vorsitzende der Gesellschaft deutscher Hals-Nasen-Ohrenärzte am offenen Grabe gesagt. "Er war der Besten einer als Mensch, Arzt und Wissenschaftler."

THE HISTORICAL BACKGROUND OF THE MODERN SPEECH CLINIC

PART 2—APHASIA

G. M. KLINGBEIL

Thucydides (*c.* —?–**399 B. C.**) the great Greek historian of antiquity, mentions that many who suffered from the plague in Athens found that, on recovery, they had not only forgotten the names of their friends and relations, but also their own names. *Hist. Lib.* **1149.**

Hippocrates* (*c.* **460-357 B. C.**) the "Father of Medicine," justly famed for crystallizing the knowledge extant into systematic science, for dissociating theurgy and philosophy from medicine, and giving the highest moral inspiration to physicians, clearly distinguished between loss of speech and loss of the voice. *De Morb. Pop.* Lib. iii, Sec. ii.

Virgil—Publius Vergilius Maro (70-19 B. C.) one of the most celebrated of Roman poets, mentions the symptoms occurring as the result of shock. *Ænid.*

Pliny (23-79 A. D.) an early Italian writer who included in his works a treatise of three books, *Studiosus*, dealing with the training of a young orator from the nursery till his entrance into public life, relates that the orator Messala Corvinus could not recall his own name. *Hist. Nat. Lib VII.*

Hoffman, Friedrich (1660-1742) a celebrated German physician, born at Halle, and later professor of medicine there, and appointed in 1708 physician to the king of Prussia, reported cases of Aphasia due to cerebral irritation from the reflex action due to worms. *Hoffmanni Opera, tom iii,* **1740.**

Gall, Franz Joseph (1757-1828) was born at Tiefenbronn in Baden. From 1796–1801 he lectured on a new theory of the function of the brain, concerning the localization of the various human faculties and proclivities in different centers. He and his pupil **Spurzheim**† in a joint treatise on phrenology in four volumes, demonstrated the crossing of the pyramids, and located the frontal lobes as the seat of word memory. The work contained the theory that the brain is a bundle of organs presiding over the different moral, sexual and intellectual traits of the individual. This theory drove him out of Vienna. He stated that full knowledge of things was compatible with inability to discover or pronounce their names. In 1819 he described two cases of aphasia and attempted to localize speech function in the anterior part of the brain. The following year he stated that the cerebral cortex was the seat of psychic functions. *Craniologie ou découvertes nouvelles du Docteur F. J. Gall.* Paris, **1807.** *Recherches sur le systeme nerveux en général, et sur celui du cerveau en particulier.—Mémoire présenté à l'Institut de France.* 1818. *Anatomie et Physiologie du Système nerveux.* **1810.**

Spurzheim, Johann Caspar (1776-1832) was a German phrenologist born near Treves, who studied medicine at Vienna, and became the pupil and later the colleague of **Gall.** He lived and lectured in France and England alternately. He worked with **Gall**† on the localisation of functions of the

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* *Vide*, Part 1.

† *Vide Infra.*

brain and co-operated with him in the publication of *Anatomie et Physiologie du Système Nerveux*. His propagandism led to the formation of secret phrenological societies and phrenological journals in Gt. Britain, and the United States. *Phrenology*, **1825.**

Crichton was one of the earliest Englishmen to interest himself in aphasia. He mentions various cases of inability to use the right word. *An Enquiry into the Nature and Origin of Mental Derangement comprehending a Concise System of the Physiology and Pathology of the Human Mind*. London, **1798.**

Baillie, Matthew (1761-1823) was a native of Lanarkshire, Scotland, who became physician to George III. His *Morbid Anatomy* published in London in 1793 is the first attempt to treat pathology as a subject in and for itself. He reported in 1813 a case of right hemiplegia in which the patient had lost recollection of nearly all words of his own language, though his pronunciation remained clear. *Medical Transactions of the College of Surgeons*, **1813.**

Lordat ascribed aphasia not to paralysis of the tongue, but to inco-ordination of the muscles used in speaking. He also recognised paraphasia, which he called "paramnésie verbale." *Rév. pér. de la Société de Paris*, **December, 1820.**

Velpeau, Alfred Armand Louis Marie* (1795-1867) cited the case of a man of great loquacity who was found to have lesion of the two anterior lobes of the brain. *Treatise on Surgical Anatomy*, 1832. *Treatise on Operative Surgery with atlas*, **1832.**

Flourens, Marie Pierre (1794-1867) a celebrated French experimental physiologist and author, a voluminous writer on human and comparative anatomy and physiology, the discoverer of the noeud vital, the bilateral center of respiration in the medulla oblongata, demonstrated, through experiments, that ths brain is the organ of thought and of will power, while the cerebellum presidee over the co-ordination of bodily movements. He held that any part of the hemisphere could carry on to a greater or less extent the general functions exercised by the whole, and if the surface of the brain was gradually sliced away, all of them would be more or less affected. He denied the possibility of any cortical localisation of function. During the second decade of the nineteenth century he emphasized the unified functioning of the cerebral lobes, and in the years 1861–1871 he excited the supporters of cerebral localisation by his denials of their theory. *Expériences sur le système nerveux*, **1825.**

Bouillaud, Jean Baptiste (1796-1881) of Angoulême, teacher of medicine and head of the Charité, was one of the ablest diagnosticians of his time. In 1825 he located the faculty of articulation in the frontal lobes of the brain, which he considered to be the organ responsible for the formation of words and of memory. He was the first to point out that aphasia is correlated with a lesion of the anterior lobes of the brain. He showed that the tongue, and correlated organs of speech, could be paralized for the act of talking, though there was no loss of power in other parts of the body. Moreover, the tongue might still be capable of all its ordinary movements, though speech was gravely affected. He held that no severe lesion of the anterior lobes of the brain could occur unaccompanied by disturbance of speech. He collected 114 observations of disease of the anterior lobes accompanied by lesion of the faculty of speech. *Recherches cliniques propres à démontrer que la perte de la parole correspond à la lesion des lobules antérieurs du cerveau. Archives Gén. de Méd.* **1825.** *Traité clinique et physiologique de l'encephalite ou inflammation du cerveau*. Paris, **1825.** *Recherches cliniques propres à demontrer que le sens du langage articulé et le principe coordinateur des mouvements de la parole résident dans les lobules antérieurs du cerveau. Bull. de l'Acad. Royale d. Méd.*, **1847-8.**

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* *Vide*, Part 1.

Bérard, Joseph Frederic (1789-1828) was a French physician and philosopher, who was a professor of medicine in Paris from 1823–1836. He reported a victim of a mine explosion who had no paralysis afterwards, and who spoke well also, but who died within 25 hours of the accident. Examination showed that the two internal thirds of the right anterior lobe, and the entire left anterior lobe were reduced to a jelly.

Jackson, S., of America, reported the case of a patient who awoke with a headache, checked cutaneous respiration and the inability to speak or write. He was bled, and speech returned, but was lost again in 15 minutes, so he was bled again, and speech returned. *Amer. Journ. of Med. Sciences.* **Feb., 1829.**

Foville, Achille, a distinguished alienist, established the fact that the cerebral cortex is the seat of psychic functions by his experimental and pathological work in **1830.**

Abercrombie, John (1780-1844) was an eminent Scottish physician who was born in Aberdeen and practiced in Edinburgh and became known as the first consulting physician in Scotland in his time. He has recorded many cases of impairment of speech, the most noteworthy being the case of a man who developed a tendency to misapply words. At the autopsy there was found to be a soft vascular cyst at the posterior part of the left hemisphere; the case of a man who developed left hemiplegia and inarticulate speech. His intelligence was unimpaired. Later there was complete loss of speech. There was found to be some softening at the outer part of the right hemisphere. And the case of a child of 4, with very imperfect articulation. Autopsy revealed cartillaginous hardening of the corpora olivaria, the crus cerebelli, and the tubercula mammillaria. *On Diseases of the Brain and Spinal Cord.*

Copland, James (1791-1870) was an English physician who studied in Edinburgh and became a member of the Royal College of Physicians. In 1822 he became editor of the *London Medical Repository.* He viewed speech disturbances as motor defects, and not as due to the loss of intellectual faculty. *On Palsy and Apoplexy,* Vol. ii.

Todd, Robert Bentley (1809-1860) was a British physician born in Dublin and educated there at Trinity College. He was professor of physiology and general and morbid anatomy at King's College, London, from 1836–1860. He recorded several instances of hemiplegia with aphasia the paralysis being always on the right side (with one exception.) *Cyclopaedia of Anatomy and Physiology,* **1836-1853.**

Romberg, Moritz Heinrich* (1795-1873) of Meiningen, who became professor of pathology in Berlin, wrote the first formal treatise on nervous diseases, making a careful collection of hitherto scattered data. He cited the case of a sailor with injury to the left side of the head, who lost power of speech for a time. He presented also another case in which partial paralysis of the left side existed along with impaired speech. A post-mortem revealed a large tumor on the right half of the pons varolii, extending posteriorly under the right olivary body. *On the Nervous Diseases of Man,* **1840-1846.**

Cruveilhier, Jean (1791-1873), of Limoges, held the first chair of pathology in the Paris Faculty, in 1836. His atlases of pathology are among the most splendidly illustrated books on the subject. He cited two cases of aphasia accompanied by degeneration of the corpus olivare.

Rochoux. At a discussion on a paper read by **Bouillaud** in 1848, he declared that he had opposed craniology since 1814, and continued to disbelieve in cerebral localisation.

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*Vide, Part 1.

Belhomme in 1848 presented a paper on the localisation of speech in which he located the memory of words in the anterior lobes of the brain.

Bergmann, of Hildesheim, in 1849 cited some well recorded cases. *Einige Bemerkungen über Störung des Gedachtness und der Sprache. Allegemeine Zeitschrift für Psychiatrie,* **1849.**

Bright, Richard (1789-1858), of Bristol, was physician at Guys Hospital, London. He had outstanding powers of observation, and was the leading consultant of his day. He reported a case of dextral paralysis with aphasia, in which there was softening of the middle and posterior lobes of the left hemisphere, the anterior lobe being unimpaired.

Hun, of Albany, cited the case of a man who was seized with the symptoms of cerebral congestion, with stupor for several days. After recovering he could understand what was said to him, but had great difficulty in expressing himself in words. He could not write the words that he could not say, though there was no paralysis. Hun concluded, therefore, that there is a portion of the brain connected with language or the memory of words as distinct from the memory of things and events, and that there is another portion of the brain on which depends the co-ordination of the movements of articulation. He noted that the impression made on the acoustic nerve was not enough for rendering the articulation of the word, but that it was necessary that an impression should be made on the optic nerve. He asked if this could be explained by the supposition of a more intimate connection between vision and articulation either by the fact that the impression on the acoustic nerve is transient while that on the optic is more permanent. *Amer. Journ. of Insanity,* **April, 1851.**

Hasbach, of Geldern, cited a case of left hemiplegia with aphasia. *Allegemeine Zeitschrift für Psychiatrie,* **1852.**

Nasse, W. In an essay on the defects of speech he cited a case of paralysis of one side of the body. In the middle of sentences this man often used the wrong words, but recognised this and tried to correct his mistakes. He stated that frequently proper names and substantives learned first in childhood seemed to disappear, but verbs and adjectives acquired later remained. *Allegemeine Zeitschrift für Psychiatrie,* **1853.**

Falret, Jean Pierre, was a French doctor who gave an original description of circular insanity in 1854. He attempted the training of idiots unsuccessfully at the Salpetrière. He collected and classified 62 cases of aphasia, but believed that greater knowledge of a detailed character was necessary before absolute conclusions were arrived at. *Des Troubles du Langage.*

Kolk, Schroeder Van Der, of Utrecht, held that there was a close connection physiologically and pathologically between the function of articulation and speech, and the corpora olivara. He cited several cases where lesion or degeneration of the olivary bodies was found to accompany impairment of speech. In 1857 he reported the case of a man in which a bony splinter from the os frontes above the left eye caused compression of the anterior part of the hemisphere with resultant loss of speech. The fragment was removed by trepan, and the speech difficulty disappeared. *Gazette Médicale de Paris,* **September 5, 1857.**

Wilks Samuel, Sir, was an English doctor associated with Guy's Hospital, London. His account of alcoholic paraplegia ranks as a classic, while his lectures on pathological anatomy in 1859 and on diseases of the nervous system in 1878 were standard sources of reference for students of his day. He held that when paralysis exists some portion of the motor tract must be affected, but that this need not arise from a local lesion of the cortical substance; hence it might be possible for hemiplegia to occur without loss of speech, and vice versa. Yet in practice he did not find this to be the case.

Brown-Séquard,* Charles Eduard (1817-1894) was a native of Mauritius who became professor of experimental medicine in the Collège de France, and later at Harvard and Paris. His outstanding work was his experiments on transactions and hemisections of the spinal cord. He stated that he had seen cases due to peripheral irritation in which no brain lesion could be discovered after death. He held that aphasia was a reflex phenomenon. *Lectures to the N. Y. Academy of Medic.*

Broca, Paul (1824-1880), the founder of modern surgery of the brain, and of the modern French school of anthropology, was, in succession, surgeon at St. Antoine, La Pitié Hôpital des Cliques and the Hôpital Neckar. He described how as a result of pathological research and histological differentiation of the various layers of the cortex it was determined that different cortical regions had specific functions, and noted the failure of observers, especially **Beaunis** and **Fournier** in France, and **Nothnagel** in Germany, to localize centers in the cortex by injecting liquids. In 1861 he "discovered" that the third left convolution of the brain was the center of articulate speech, and this theory led to the mapping out of the brain into different centers, for surgical operation. He thus originated modern methods of craniology. He differentiated between the faculty of articulated language and the general faculty of language which is the power of establishing a constant relation between an idea and a sign. He subsequently extended the area of speech to the Isle of Real and to the extra-ventricular nucleus of the corpus striatum. In 1868 he divided disorders of speech due to central lesions into "aphémie" in which the patient has a profoundly reduced vocabulary, but retains intact his ideas, and amnésie, where the patient can no longer recognise the conventional associations established between ideas and words.

Gratiolet. At a meeting of the Société Anthropologic in 1861 said, "I agree that intelligence is one, that the brain is one, and acts as a whole; but this does not exclude the idea that certain faculties of the mind stand in special relation with certain cerebral regions."

Aubertin insisted at a meeting of the Société Anthropologic in 1861 that the higher functions of the brain stand in relation to the development of the anterior lobes, especially speech. *Considérations sur les localisations cérébrales et en particulier sur le siège de la faculté du langage articulé. Gaz. Hebdom.* **1863.**

Voisin reported the case of a person with left sided hemiplegia who had no difficulty in interpreting and using language. Later when right hemiplegia set in he developed complete aphasia. *Observation de Perte de la Parole. Bulletin de l'Académie de Médicine,* **1862.**

Parrot reported a case in 1863 in which there was complete atrophy of the Isle of Reil and of the three frontal convolutions on the right side, with, nevertheless, preservation of intelligence and articulate language.

Dax, G., presented a Mémoire to the Académie de Médecine in 1864 in which he pointed out that the left half of the brain had functions with which the right was not endowed. The following year he attributed aphasia to lesion of the brain more especially on the anterior and external part of the middle lobe. *Sur le Même Sujet. Gaz. Heb.* **1865.**

Jackson, Hughlings (1834-1911) was a leading neurologist from Yorkshire, England, who did much to establish the use of the ophthalmoscope in diagnosis of brain diseases, described Jacksonian epilepsy, and originated the doctrine of levels in the nervous system. In 1864 he gave his clinical lecture on "Loss of Speech" in which he departed from the usual approach of the day which was structural, and adopted instead a dynamic conception.

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* *Vide.* Part 1.

He distinguished two main groups in the first of which he placed those who were speechless, or whose speech was very much damaged. In the second group he placed those whose speech was plentiful but disturbed by many errors. He believed that the nervous system showed a hierarchy of functional levels with successively higher levels developing later and breaking down sooner. He pointed out that destruction of functions at more highly developed levels removed inhibitions over lower centers which therefore became more easily dischargeable. This breakdown of higher centers with the removed inhibitions was to be clearly seen, he pointed out, in the phenomenon of aphasia, the damage being most in the corpus striatum. The nearer the disease is to the corpus striatum the more likely will be defect of articulation. And, conversely, the farther off, the more likely is it to be one of mistakes of words. He held also, that the phenomena of speech cases are primarily psychical, and only in the second place susceptible of physiological or anatomical explanation. The phenomena of aphasia, he recognised, never stands alone, but are accompanied by a loss of certain voluntary actions, in some cases with conservation of the more automatic. He emphasized that speech, apart from its articulatory aspect, is double—intellectual and emotional, and he saw that speechlessness could exist without imperception, and that the mind could be affected, not only on its emissory, but also on its receptive side. He declared that, to locate the damage which destroys speech, and to localize speech are two different things, though he believed that the lesion was always in the region of the corpus striatum, and that the automatic services of words were associated with the activities of the right hemisphere, except in the case of left-handed people. The lower, more automatic activities were centered in both hemispheres. Writing, he held to be affected, not as a separate faculty, but as a part of the failure to propositionalise in words. There is no such condition as pure agraphia, he affirmed, for if imperception (agnosia) is added to such difficulties of speech, the patient will show signs of inferior comprehension. He recognised also the phenomenon later known as apraxia. He is generally credited as being the first to realize the value of the psychological study of aphasia, and the primary importance of exact clinical findings. He has given details of 34 cases of hemiplegia with loss of speech, with paralysis on the right side in 31 of these cases. In an article in *Reynold's System of Medicine* he says that defects of speech with symptoms pointing to disease of the left hemisphere are not as striking in children as in adults. *On the Evolution and Dissolution of the Nervous System. The Croonian Lectures*, **1884.** *London Hospital Reports*, **1864.**

Trousseau, Armand (1801-1867), of Tours, and master of clinical delineation held the position of physician at the Hopital St. Antoine in 1839, and at Hôtel Dieu in 1850, and professor of the Paris faculty the same year. He was the first to perform tracheotomy in Paris, and was awarded the Grand Prize of the Academy of Medicine for his *Traité pratique de la phthisie laryngée*. He made aphasia a prominent subject in his clinical lectures at the Hôtel Dieu. He replaced the term aphemia by aphasia, which he divided into three divisions, which Head says bear no relation to any fundamental principles. He declared that aphasia is produced in nearly all cases by injury to the frontal lobes, the lesion being almost exclusively confined to the left hemisphere, while its most frequent situation is the posterior part of the third left convolution. He noted that of 134 observations, 124 confirmed **Dax's** left hemisphere theory, and 10 were to the contrary; while as regards **Broca's** theory that the lesion is of the third frontal convolution, 14 cases were in favor, and 18 against. *De l'Aphasie: maladie décrite récemment sous le nom impropre d'aphémie. Gaz. des Hôp.*, **1864.** *Clinique Médicale, tom 2*, **1867.**

Steward recorded the case of a woman with left hemiplegia without loss of speech, followed by paralysis of the right side with loss of speech. *Medical Times*, **July 9, 1864.**

Lesur recorded the case of a child who was trepanned about 1¼ inches above the left orbit. During the process of treatment, pressure on the brain at the exposed part suspended the power of speech, which however returned as the pressure was removed. *Gazette des Hôpitaux*, **1865.**

Ladame stated that his findings were that tumors of the corpus striatum and of the pons variolii are more frequently attended by loss of speech than those occurring in any other part of the encephalon. Tumors in the middle lobes are also more frequently accompanied by difficulties of speech than those occupying the anterior lobes in the proportion of 5 : 4. He concluded that derangement of speech was not common in cerebral tumors, since only 44 out of 332 cases observed showed it. *Gaz. des Hôp.*, **April 29, 1865.**

Gairdner, William Tennant (Sir) was born and educated at Edinburgh and became professor of medicine at Glasgow University from 1862–1900. He insisted that a person with motor aphasia writes at least as badly as he speaks, and that a person wholly unable to speak cannot write. He cited the case of a person suffering from epileptic seizure with a form of cateleptic state without coma or evident paralysis. Recovery of intelligence ensued, but the patient could not express himself in writing though he could copy writing. The necropsy revealed no cerebral lesion beyond general and diffused congestion of the vessels of the pia mater. *Glasgow Medic. Journ.*, **May, 1866.** *Proceedings of the Glascow Philos. Soc.*, **1865-1868.** *On the Function of Articulate Speech and its Connection with the Hand and Bodily Organs.* **1866.** *Aphasia and Agraphia—St. Geo. Hosp. Rep.*, Vol. 2, **1871.**

Dax, Marc, of Sommières, and father of **G. Dax,*** stated as a result of observation of 140 cases that patients suffering from aphasia along with paralysis, were always paralysed on the right side. He concluded therefore that the faculty of language was situated on the left side of the brain. *Lésions de la moitié gauche de l'encéphale cöincédant avec l'oublie des signes de la pensée. Gaz. Hebdom.*, **1865.**

Benedikt, Moriz
Braunwart } In a paper reported by **Heymann** they cited examples of aphasia and agraphia resulting from acute lead poisoning. *Canstates Jahresbuch*, **1865.**

Banks, who was a professor of Trinity College, reported a man with paralysis of the right side with accompanying aphasia. Loss of speech was complete at first, but after two days he could speak a little, and he continued to improve. He therefore concluded that, even with aphasia with paralysis, the mind may be unclouded, and the power of memory be restored, even after years. *Clinical Medicine*, **1865.**

Flaubert reported in 1866 the case of a patient in Rouen with a skull fracture but with no symptoms of either paralysis or aphasia, though, in the right hemisphere, the anterior and inferior extremity of the frontal lobe was destroyed, softened and infiltrated with blood. The three frontal convolutions were affected, the third only in its anterior half. In the left hemisphere there was complete destruction of the frontal lobe as far as the transverse frontal convolution, a pulpy mass only remaining. The convolution of the Isle of Reil was flattened in both hemispheres. *Font-Réaulx—Localisation de la faculté du gang. articulé.* Paris, **1866.**

Flint, Austin, gave an account of six cases under his observation. He expressed strong dissent from the doctrine of the localisation of the faculty of speech in the left hemisphere.

Sanders reported in 1866 a case of incomplete paralysis of the right side, with defective speech and loss of memory of words. There was lesion of the left hemisphere. The posterior part of the inferior left frontal convolution,

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* *Vide supra.*

where it forms the anterior margin of the fissure of Sylvius, was damaged, and there was also softening of the posterior extremity of the fissure of Sylvius. He also stated his belief that in speech palsy it is the Isle of Reil rather than Broca's convolution that is at fault. *Articles in the Lancet for* **June, 1866.** *Articles in the Edinburgh Medic. Journ.*, **Aug., 1866.**

Simpson, James Young (Sir), (1811-1870) was a Scottish physician from Bathgate, who became professor of obstetrics at Edinburgh, 1840. By his statistical investigation of the results of major operations he did much to improve hospital administration. He quoted in 1867 a case of extensive lesion of the third frontal convolution without aphasia. *Medical Times*, **Dec. 21, 1867.**

Robertson, John, after citing three interesting cases of aphasia, summarized his views as follows: "There is usually lesion of the left hemisphere of the brain, of efferent fibres passing between the convolutions and the great co-ordinating centers, probably at some point of a line extending from the external frontal convolution to the corpus striatum, so that the voluntary motor impulses for the articulation of language cannot be transmitted. The essential morbid change is therefore motor, not mental." He was a Scottish doctor from Glasgow. *The Pathology of Aphasia, Journ. of Mental Sc.*, **Jan., 1867.**

Ogle, John Wilhelm, was the first to use the word agraphia, although the condition was earlier described by **Marcé.** He considered that the occasional separation of agraphia and aphasia to be an argument in favor of distinct cerebral centers for the faculties concerned in speech and writing, while the more frequent coincidence of the two would lead to the inference that these distinct centers must be closely contiguous. *Aphasia and Agraphia. St. George's Hospital Reports*, Vol. 2, **1867.**

Lyons reported a boy with cerebro spinal arachnitis, followed by aphasia with right hemiplegia. He regained speech for two months, but relapsed and was aphasic to his death. *Brit. Med. Journ.*, **Sept., 1867.**

Jackson, Scoresby cited the case of a patient with a wound to the left temple followed by right hemiplegia, and some time later with aphasia. At his subsequent death it was found that there was considerable softening of the left hemisphere. This was most advanced in the Isle of Reil and in the posterior part of the third frontal convolution. *Edin. Medic. Journ.*, **Feb., 1867.**

Jaccoud, Francois Sigismond (1830-1913) was a French physician and professor of pathology in the Paris Faculty of Medicine, permanent secretary of the Académie de Medicine from 1877, and a teacher in the medical clinic at the Pitié from 1883. His treatise on medical practice in 1871 and his clinical lectures, 1867–1888, enjoyed great reputation. In 1867 he reported a case which had become aphasic for five weeks and then spoke again. There was well defined softening of the white substance in the neighborhood of the third frontal convolution of the left anterior lobe, though the convolution itself was not affected. *Gaz. des Hôp.*, **May 16, 1867.**

Castagnon reported in 1867 a case of traumatic aphasia resulting from a shot on the head which penetrated the antero superior portion of the left parietal. *Gaz. des Hôp.*, **Oct. 12, 1867.**

Blake, Paget, cited a case of poisoning by stramonium in which speech was lost entirely at first, later everything was misnamed. *St. Geo. Hosp. Reports*, **1868.**

Vulpian, E. F. A., who helped to found the "Archives de Physiologie normale et physiologique," reported a case of loss of power of speech on admission to hospital, who later became hemiplegic on the right side. Softening in the posterior half of the supra ventricular white matter of the left hemisphere was found, but no indication of lesion of the frontal or other convolutions.

Westphal, Carl Friedrich Otto (1833-1890) was a German alienist, born in Berlin, who studied medicine at Berlin, Heidelberg and Zurich. He became professor of psychiatrics in the University of Berlin from 1874–1890. In 1868 he became editor of the *Archiv für Psychiatrie und Nervenkrankheiten.* He described agoraphobia and pseudosclerosis, and pointed out the importance of the knee jerk in diagnosis. He reported the case of a patient who showed loss of appreciation of posture and passive movement. This he attributed to loss of kinaesthetic memory.

Seguin, Edward Constant (1843-1898) was an American neurologist born in Paris, France. He attended the College of Physicians and Surgeons in New York City, then studied for a year under **Brown-Séquard,** and **Charcot** in Paris. He became lecturer and then professor of nervous diseases in New York, and founded the clinic for nervous diseases in New York in 1873. He was the editor of the *American Series of Clinical Lectures.* He collected details of 556 cases of aphasia from various sources and tabulated them according to the parts of the encephalon damaged. Out of 270 cases of hemiplegia with aphasia he found that 243 were paralysed on the right side of the body, and 17 on the left. He stated that this was the ratio of left handed to right handed people. *A Statement of the Aphasic Question together with a Report of 50 cases.—Am. Quart. Journ. of Psychol. Medic.,* **Jan., 1868.** *Opera Minora,* **1884.**

Bastian, Henry Charlton (1837-1915), of Truro, was one of the founders of English neurology, and the author of important books on the brain, the paralyses, aphasia, and spontaneous generation. In 1867 he reported a case of right hemiplegia with aphasia in which the arteries at the base of the brain were found to be notably diseased. In 1869 he published his famous paper. Here he declared that words become nascent as revived auditory impressions. He divided the higher forms of speech disorders into amnesia, and aphasia proper. He affirmed that thinking power is impaired in almost direct proportion to the loss of the power of expression. In amnesia the patient suffered a loss of words, speech and thought. But the aphasic patient retained the power of thought though he had great difficulty in expressing himself in speaking or writing. He held that all high grade disorders of speech were due either to the destruction of the auditory and visual centers, or to some affection of the fibres transmitting impressions between them and the lower motor mechanism for the tongue and lips or of the hand for writing. He located the visual and auditory word centers, and was the first to describe word-deafness and word-blindness, and to recognise the relation of defective auditory perception to speech production. He introduced the term "jargon-aphasia." *On the Various Forms of Loss of Speech in Cerebral Disease. Brit. & For. Med. Chi. Rev.,* **1869.** *The Psychology of Thinking. Fortnightly Rev.,* **1869.** *On Some Problems in Connection with Aphasia and Other Speech Defects. The Lumelian Lectures. Lancet,* **1897.** *Aphasia and Other Speech Defects.* London, **1898.**

Béhier, a professor at Hotel Dieu, reported in 1869 the case of a woman with right hemiplegia whose aphasia took the form of ability merely to repeat words said in her presence, without attaching any meaning to them. He also collected 122 observations opposed to the theories of **MM. Bouillaud, Dax** and **Broca;** in 82 of these cases lesion of the anterior lobes had been observed without aphasia, and in 34 cases aphasia co-incided with disease of other parts of the brain. *Gaz. des Hôp.,* **March 20, 1869.**

Smith, Franks, declared that hammer palsy might cause loss of speech through central vibration, without visible lesion, by simple molicular disturbance of the brain cells. *Lancet,* **March 27, 1869.**

Finkelnburg, F. C. M., held that disorders of speech are part of wider disturbances called symbollic representation. There is a more or less profound disturbance of the power to receive or impart knowledge as far as this depends on

sensory symbols. The condition is both motor and sensory, and an organic lesion must disturb both aspects of function. *Niederrhanische Gesellschaft, Sitzung vom März,* **1870.** Bonn.

Hitzig, Eduard (1838-1907) was born in Berlin and studied medicine there and in Würzburg. He became professor of psychiatry in Zurich in 1875, and in Halle in 1879, and there he established the first clinic in Prussia for nervous and mental disorders. He was especially interested in the localisation of the various functions. He and **Fritsch,** through their experiments on the cortex of animals proved by electrical stimulation the presence of motor areas. *Physiologische und klinische Untersuchungen über das Gehirn,* **1904.**

Hammond, William Alexander (1828-1900) was an American neurologist, born in Annapolis, Md. He was professor of mental and nervous diseases at Bellevue Hospital Medical College, 1867–1873, and occupied a similar chair in New York University, 1873–1882. He helped found the New York Post-graduate Medical School and established the *Quarterly Journal of Psychological Medicine and Medical Jurisprudence.* Regarding defects of speech he rejected the restricted location of the **MM. Dax** and the still more limited area of **M. Broca.** He held that the organ of language is situated in both hemispheres and in that part which is nourished by the middle cerebral artery. He declared that while the more frequent occurrence of right hemiplegia in connection with aphasia is in great part the result of anatomical arrangements of the arteries which favor embolism on that side, there is strong evidence to show that the left side of the brain is more intimately connected with the faculty of speech than the right. *Diseases of the Nervous System,* **1871.**

Batty, Tuke } Gave a case in 1872, in which the posterior half of the third
Frazer, John } frontal convolution was completely destroyed both as regards white and gray matter, but the only defect in language was partial verbal amnesia. There was no right hemiplegia. The doctors in case believed that this proves Broca's theory to be wrong, but **Dr. Wilks** believed that it corroborated Broca's views. *Journal of Medical Science,* **July, 1872.**

Broadbent, William Henry (Sir), (1835-1907) was educated at the Royal School of Medicine in Manchester, and in Paris. In 1881 he became president of the Medical Society of London. From 1898–1901 he was physician extraordinary to Queen Victoria, and later physician in ordinary to Edward VII. He mapped out portions of the cortex which he said must contain the perception centers for hearing and vision, and he conceived of a propositionizing center on the motor side of the brain and of a naming center on the sensory side. He deduced clinical modalities of aphasia from suppositious lesions of the speech centers or their commisural paths, and pointed out the existence of word blindness. *On the Cerebral Mechanism of Speech and Thought. Med. Chi. Trans.,* **1872.** *A Case of Peculiar Affection of Speech with Commentary. Brain,* **1879.**

Proust, Adrian. Bateman declared that his essay written in 1872 entitled *De l'Aphasie* was lucid and incisive.

Ferrier, David (Sir), (1843-?) carried on experimental researches on mammals, birds, frogs and fish, and by his work with the faradic current he was able to confirm the findings of **Hitzig** and **Fritsch.*** At the International Medical Congress in 1881 he produced a dog which had the superior temporo-sphenoidal convolution destroyed, with the result that the animal had been made deaf. His experimental researches led him to place the auditory center in the first temporal convolution, and that for vision in the supra marginal and angular gyri. To Broca's area he assigned the glosso kinaesthetic center, and placed writing in the posterior portion of the second

**Vide supra.*

frontal convolution. *Experimental Researches in Cerebral Physiology and Pathology. West Riding Asylum Reports,* **1873.** *Experiments on the Brain of Monkeys. Phil. Trans. Roy. Soc.,* **1875.**

Wernicke, Carl (1848-1905) came from Upper Silesia and was professor at Berlin in 1885, at Breslau in 1890 and was particularly interested in brain diseases and insanity. He issued a splendid atlas of the brain. His work, *Der Aphasische Symptomencomplex,* was the outcome of the researches of **Meynerts** on the projection systems of the cortex. He declared that the posterior part of the brain was sensory, and the anterior part was motor. However, since the cerebrum is essentially the organ of consciousness, these motor centers must be occupied with conception of movement. He placed the audition center, the seat of sound memory, in the first temporal convolution, and the conceptional basis of speech, in Broca's area. Destruction of the first temporal gyrus, he declared, abolished sound images, and resulted therefore in lack of understanding of spoken words. Speech was free and plentiful, but characterized by errors. Furthermore there were defects of writing and reading. This syndrome was the famous sensory aphasia, known now as Wernicke's aphasia. Motor aphasia, he held, resulted from the destruction of the third frontal convolution; conduction aphasia was due to interruption of the path between the two centers; the result here is that words are misapplied, but understanding is complete. In total aphasia there is lesion of both centers. The term sensory aphasia replaced the older term, amnesic aphasia. *Der Aphasische Symptomencomplex.* Breslau, **1874.** *Lehrbuch der Gehirnkrankheiten Kassel,* **1881.** *Ein Fall von isolierter Agraphie Monatschr. f. Psychiat. u. Neurol.,* **1903.**

Charcot, Jean Martin (1825-1893) was a French physician at Salpêtrière where he created the greatest neurological clinic of modern times, according to Garrison. He based his explanation of aphasia on a psychological analysis of speech, contrasting the acquisition of speech with the phenomenon of dissociation occurring in disease. He reported the case of a woman with left hemiplegia, with yellow softening of the surface of the right frontal lobe, the second and third frontal convolutions being completely destroyed, yet who had no embarrassment of speech or loss of memory. He also produced 10 or 12 cases confirming Broca's theory and gave a case where Broca's convolution was intact, but where there was a lesion in the posterior part of the Isle of Reil and the lower marginal convolution. *Cécité Verbale,* **1887.** *Leçons sur les Localisations dans les Maladies du Cerveau, receuillis par Bourneville,* **1876.**

Kussmaul, Adolf (1822-1902) was born near Graben, and educated at Heidelberg. In 1857 he became professor and director of the clinic at Heidelberg, and afterwards held the chair at Erlangen from 1859–1863, at Freidburg from 1863–1876, and at Straszburg from 1876–1889. He regarded speech as an organized reflex. All verbal expression, he declared, follows three stages: preparation, internal diction, and articulation. He believed, therefore, that you could not seek a seat of speech in the brain. Local lesions must be associated with some particular damage to a complex group of ganglionic mechanism. Visual and auditory images may remain unaffected, though their symbolic significance can no longer be recognised. He affirmed that word blindness and word deafness are essentially disorders independent of defects of speech. Word blindness had been described by **Broadbent*** and **Wernicke*** but had never been clearly differentiated until Kussmaul did it. *Die Störung der Sprache. Ziemssen's Handbuch d. speciellen Pathologie u. Therapie,* **1877.**

Barlow presented a case to show that, though the motor speech center in Broca's area might be destroyed, the corresponding part of the right hemisphere might take up its function. *Brit. Med. Journ.,* Vol. 1, **1877.**

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* *Vide supra.*

Munk, Hermann (1839-1912) was a German physiologist born at Posen, who studied at Berlin and Göttingen and who, in 1862, became docent in Berlin University, and later professor of physiology at the veterinary college at Berlin. He was particularly interested in the physiology of the nerves and the brain. He determined the true position of the visual center in the occipital lobes and added the conception of mind blindedness. *Über die Funktionen der Grosshirnrinde*, **1881.** *Über die Ausdehnung der Sinnesphären in der Grosshirnrinde*, **1901.** *Zür Anatomie und Physiologie der Sehspare der Grosshirnrinde*, **1910.**

Foulis, of Glasgow, reported not the least symptom of speech defect in a patient with cardiac dropsy, except a tendency to lie in bed and conduct imaginary narratives in a casual sort of way and to refer to events as presently occurring, which had no existence except in his imagination. The third left frontal convolutions were almost completely destroyed along with two-thirds of the Isle of Reil. The patient was right-handed, and there was no hemiplegia, and aphasia was at no time a feature of the case though the destruction of the third left convolution was so complete as to abolish any function which it may have exercised. He believed this case disproved Broca's theory. *Brit. Med. Journ.*, **March 15, 1879.**

Gowers, William Richard (1845-1915), of London, was well known for his treatises on the spinal cord, diseases of the brain, and the nervous system, which did much to systematize existing knowledge of these conditions. Speaking of the loss of speech in certain cases of tubercular meningitis, he said that it was probably a consequence of irritative inhibitions, and that the speech center seemed specially susceptible of inhibitions. *Diseases of the Nervous System*, **1886-1888.**

Bigelow, Henry Jacob (1816-1890), of Boston, Mass., became surgeon to the Massachusetts General Hospital and professor of surgery, and the leading surgeon of New England during his life time. He reported a case of very extensive injury to the brain without loss of power of articulated language. An explosive drove an iron through the face of the patient, from the jaw up on the left side, slightly in; it emerged in the neighborhood of the anterior fontenelle. The patient remained in full power of speech though he became, afterwards, very profane, something he had never been before the accident.

Goltz Friedrich Leopold (1834-1902) was a German physiologist born at Posen He became professor at Königsberg in 1865, and professor of physiology at Halle in 1870 and at Straszburg in 1872. He was the chief of the few remaining champions of **Flourens'** system. His most telling experiments were those on the effects of excision of the brain and spinal cord of the frog and dog. He showed that the effects of decerebration are the more profound the higher the animal. He exhibited a dog which he had operated on as a practical refutation of the theory of localisation. However, the postmortem later revealed that the cortical areas had not been destroyed as completely as Goltz had believed. He asserted that the same disturbance of motion, sensation, and vision follows extirpation of any part of the hemisphere, the more extensive the destruction, the graver the loss of function. He did not believe in the theory of specific cortical centers. *Trans. International Medic. Congress*, **1881.**

Grattan, Nicholas, reported in 1881 a case of aphasia following belladonna poisoning. Speech was restored when the effects of the drug were counteracted. *Lancet*, **1881.**

Exner, Siegmund (1846-1926) was an Austrian physiologist born at Vienna, who studied at Heidelberg, and lectured at Vienna University and became professor of physiology in 1875. He made many investigations into the physiology of the nervous system. He allocated a separated area of the brain for writing—the left second frontal convolution. *Untersuchung über die Localisation der Functionen in der Groszhirnrinde des Menschen.* Wien, **1881.**

Skwortzoff, Mme. Nadine, made an analysis of many cases of sensory aphasia which portrayed the teachings of **Magnan.** She contributed valuable observations of cases of word blindness and word deafness. *De la Cécité et de la Surdité des Mots dans l'Aphasie,* **1881.**

Spitzka, Edward Charles (1852-1914) was an American psychiatrist born in New York City, and educated in New York University, Leipzig and Vienna. He served as interne in the New York City Asylum for the Insane and was professor of medical jurisprudence and diseases of the nervous system in the New York Post-Graduate Medical School and Hospital, 1885–1887, and was editor of the *American Journal of Neurology,* 1881–1884, and in 1890 president of the American Neurological Society. He did not consider that exaggeration of the tendon reflex was significant in cases of aphasia. He reported 8 cases of exaggerated knee jerk of which only one had any marked speech affection, and in two cases in which the reflex was abolished, speech was very much affected. *Am. Journ. of Neurology and Psychol.,* **Aug., 1883.**

Pitres argued in 1884 for the existence of a graphic center, and in 1895 confirmed the theory that the motor area of the cerebral cortex is the central sulcus of Rolando.

Lichtheim, Ludwig, drew a triangle, the angles of which represented the three speech centers, visual, auditory, and kinaesthetic, and thus showed that no less than 14 different areas might be the seat of a lesion producing aphasia. He gave seven forms of disorders of speech. *On Aphasia.* Brain, **1885.**

Stewart, Grainger, gave an excellent resumé of the topic aphasia in his book, according to **Elder,** *An Introduction to the Study of Diseases of the Nervous System,* **1885.**

Amidon made a contribution to the pathological anatomy of sensory aphasia which included a critical analysis of several typical cases reported by European writers. *On the Pathological Anatomy of Sensory Aphasia. New York Journ.,* **Jan., 1885.**

Bernard, Claude (1813-1878), the greatest physiologist of modern France and the founder of experimental medicine, gave a course of lectures at the College of France and the Sorbonne, including lectures on the physiology and pathology of the nervous system. His work on aphasia ranks next to **Kussmaul's*** as the most complete treatise on the subject. He was a disciple of **Charcot.***

Déjerine, Jules (1849-1918) of Geneva, took his M.D. at Paris and was successively chief of clinics at Bicêtre, 1882–1891, professor of the history of medicine in the Paris Faculty, and clinic chief at Salpêtrière, 1911–1917. He married an American who collaborated with him in his great work on the anatomy of the nervous system. He defended the assumption of motor and sensory aphasia, and a zone of language containing motor and sensory images of words. Defects of intelligence in all aphasias of cortical origin, he declared to be due to the suppression of language images. Sub-cortical lesions, he said, produce pure aphasia, in which intelligence remains intact In collaboration with **Paul Sérieux**† in 1891 and 1892, he proved the existence of two distinct forms of word-blindness, produced by lesions in two situations, and at the same time, tried to disprove the existence of agraphia pure and simple, as an independent form of aphasia. *L'Aphasie Sensorielle et l'Aphasie Motrice. Semiologie des Affections du Systeme Nerveux.*

Sérieux, Paul (1864-?), who wrote on "Reasoned Insanity," worked with **Déjerine**† in publication of *Compte Rendu de la Société Biologique* during 1891 and 1892, to prove the existence of two distinct forms of word-blindness.

Rieger who was professor of psychiatry at Würzburg, constructed a "true battery of tests for the study of the aphasic patient," publishing it in 1888.

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* *Vide infra.*
† *Vide supra.*

Henschen, S. E., was interested in the word-hearing and visual centers, and helped map out the sensory and motor areas of the brain, and made many valuable observations on the pathological localisation of aphasic lesions. He took the records of every case of aphasia sensory or motor, together with their subsidiary forms, which contained a sufficiently detailed description of the post-mortem descriptions. He then tabulated every group of anatomical lesions, with the recorded symptoms, in order to find some correlation between them. He concluded that within the left temporal lobe, there are two sharply defined centers, one of which in the gyrus transversus, is responsible for the auditory comprehension of words. These two centers are anatomically and functionally separable. Speech deafness is the inability to understand the spoken word. Word-blindness caused by destruction of the tissue of the angular gyrus, consists of inability to read words. It may include inability to appreciate the significance of the object, or may be confined to words or letters. In addition to these varieties of sensory aphasia Henschen concluded that the so-called transcortical form, due to lesions situated between the principal groups of centers, seems to stand on an assured basis. Lesion of the cortex in the third frontal region produces a want of power of co-ordination of letters and syllables to form words. The patient knows what he wants to say but cannot find suitable forms of verbal expressions. He suffers from aphemia, for he has forgotten the movements of speech. Anarthria or disarthria he defined as failure because of defective vocalisation, not from lack of vocal memory. *Pathologie des Gehirns.* Upsala, **1890.** *Ueber die Hörsphäre. Journ. f. Psycholog. u. Neurol.*, **1918.**

Wyllie gave in 1894 a very accurate and comprehensive account, especially of the contribution of the French school in regard to word blindness. *Disorders of Speech*, **1894.**

Hinshelwood added several important observations in an article published in the *Lancet*, **Dec., 1895.**

Déjerine, Mme. Augustine (—**?-1901**) näe Klumpke, of San Francisco, a Paris graduate of 1889, collaborated with her husband **Jules** in his great work on the anatomy of the nervous centers. She gave a convincing demonstration on the constitution of the quadrilateral area and pointed out that any lesion of its anterior and external parts must contain fibres from the third frontal convolution. Motor aphasia, therefore, was dependent on destruction, cortical or subcortical, of fibres issuing from the front portion of the zone of language; sensory aphasia, however, was due to a lesion of Wernicke's area.

Freund, of Breslau, had a narrow conception of so-called sensory aphasia, according to Weisenberg and McBride. *Labyrenthtaubheit und Sprachtaubheit.* Wiesbaden, **1895.**

Pick, Arnold, was particularly interested in agrammatism in which the syntax of the sentence is more particularly affected. It is the result of a disturbance of purposive and orderly procedure—the use of language. He separated the formulation of thought, which is a preliminary to the use of language, from the formulation of speech, which is a necessary part of the executive process. He declared that there was a psychological schema which comprised the thought, which was distinct from the grammatical schema, which preceded its formation in words. He laid stress on the lack of inhibition as an important factor in the production of this variety of disordered speech. He insisted that we are not dealing here with the loss of independent psychical- but with disturbance of a highly organised-process. He also emphasised the close relationship between apraxia and asymbolia. *Beiträge zur Pathologie und pathologischen Anatomie des Zentralnervensystems.* Berlin, **1898.** *Studien über motorische Aprazie.* Leipzig, **1905.** *Die Agrammatischen Sprachstörungen.* Berlin, **1913.**

Winslow, L. S. Forbes (1844-1913) was the editor of the *Psychological Journal* for eight years and the founder of the British Hospital for Mental Disorders. He wrote treatises dealing with the legal and picturesque aspects of lunacy. In his work on the brain he went into considerable detail on the question

of the cerebral localisation of speech, and gave many cases. In 54 of them he detected after death a considerable amount of disease of the anterior lobe, often unaccompanied, during life, with loss of speech, though sometimes speech was affected. *Clinical Diseases of the Brain*, **1898.**

Liepmann, Hugo Carl, suggested that all changes in speech and motor behavior could be included under the term apraxia. Five years later, in 1905, he said that apraxia could be called the aphasia of the extremities, or that expressive aphasia could be called apraxia of the speech muscles. He tried to classify disorders of speech as forms of apraxia and agnosia. Apraxia he defined as inability to act or move various parts of the body in a purposeful manner though the power of movement is intact. There is no paralysis, and the patient understands what is said to him, and what he wants to say, but cannot find the means of expression. He classified the various forms of apraxia as: limb kinetic apraxia, which is due to uncomplicated loss of kinesthetic memories; ideokinetic apraxia, in which the kinetic mechanism of the limb is intact, but is dissociated from the total conception of its purposive movements; here the ideational and kinesthetic processes are separated from each other and we have motor apraxia in the full sense of the term; and thirdly, ideational apraxia in which the mechanical kinesis of the limb and the conception of the movements are intact, but the primary or initiating idea is at fault. He held that anarthria and apraxia must be distinguished from each other, but that in principle, physiologically and psychologically, the expressive, aphasic disturbances, belong to the sphere of apraxia. *Das Krankheitbild der Apraxie.* Berlin, **1900.** *Fall von linksseitiger Agraphie und Apraxie bei rechtsseiliger Lahmung. Journ. f. Psychol. u. Neurol.*, Vol. 10, **1908.**

Mills, Charles Karsner (1845-?) was an American neurologist born in Philadelphia and educated in the University of Pennsylvania. He was professor of diseases of the mind and nervous system at the Philadelphia Polyclinic, 1883–1898, clinical professor of nervous diseases at the Women's Medical College of Pennsylvania, professor of mental diseases and medical jurisprudence, 1893–1901, clinical professor of nervous diseases, 1901–1903, and professor of neurology from 1903 in the University of Pennsylvania. He differentiated a motor center in the hinder part of the third frontal convolution and extending to the insula; an auditory speech center in the first and second temporal gyri; visual centers in the angular gyrus; a graphic center in the caudal half of the second frontal, and a naming center in the three temporal gyri. *Aphasia and the Cerebral Zone of Speech*, **1904.**

Marie, Pierre (1853-?) was **Charcot's**‡ ablest pupil. He graduated in 1883 and became professor in the Paris Faculty in 1809. He presented in 1906 three papers in the Semaine Médicale on *Revision of the Question of Aphasia*, in which he denied that the three frontal convolutions played any part in the function of language. He recognised no distinction between motor and sensory aphasias and declared that the left temporo-parietal lobe was a region of intelligence specialized for language, and not solely a center of sensory images. Every true aphasic, he held, shows some want of comprehension of spoken words, therefore a graduated test is necessary. In every case, intellectual capability is diminished as a whole. Anarthria, he said, is loss of control of all those complex mechanical aptitudes which are employed in the exteriorisation of language. It can exist alone without aphasia, and if so, internal speech is intact. The patient can understand what is said to him, and can read and write. He declared that there was only one true aphasia, which he allowed was caused by lesions in the region of Wernicke's zone. Whenever this is destroyed the loss of capacity extends over a wide range of intellectual functions. The only other types of aphasia which existed for him were anarthria and pure alexia, which were due, according to him, to subcortical lesion. *La Trosième circonvolution frontale gauche ne joue aucun rôle spécial dans la fonction de langage. Semaine Médicale*, **1906.** *L'Aphasie de* 1861–1866. *Semaine Médicale*, **1906.** *Ueber den Gegenwartigen Stand der Frage nach der Localization im Grosshirn Aphasie, Agnosie, Apraxie. Ergebuisse der Psychologie*, Vol. 6, **1907.**

‡ *Vide supra.*

Monakow, Constantin von, helped to map out the motor and sensory areas of the brain. He had a theory of loss of functional continuity at the synapses. He insisted that the nervous system acted as a whole, so that if a lesion in the motor speech area occurs, you would get a depression of function in the visual, auditory, and other speech areas which would thus cause aphasia. The signs of apraxia and agnosia were likely to appear together with aphasia as part of the picture. He propounded a doctrine of diachisis, and showed that aphasia and amnesia, like apraxia and agnosia were temporary disorders of function which tended to diminish greatly, if the lesion remained stationary, but that acute exacerbation of the disease might produce manifestations far exceeding in severity and extent any loss of function that might be attributed directly to the injured portion of the brain. Therefore you cannot establish a strict and detailed relation between the site of the anatomical lesion and the specific forms assumed by the defects of speech. Local symptoms which follow non-progressive lesion are severer, more extensive, and often less sharply determined than is the case after the expiration of some days or weeks. The initial manifestations may differ profoundly in character from the residual consequence of irreparable anatomical destruction. He defined diaschisis as a suspension of activity which usually arises suddenly affecting a widely radiating central field of function. It takes its origin in the local lesion and does not attack the whole cortex. The loss of irritability and cessation of function spreads into neighboring and closely allied neuronal systems. Restoration of function depends on recovery from pathological processes of a grossly organic nature; the passing away of diaschisis and the reappearance of normal activity at synaptic junctions; the gradual assumption later of compensatory powers by uninjured portions of the nervous system. Aphasia must be considered not only as direct manifestation of the physiological activity of the damaged area, but also from the point of view of the acuteness and duration of the lesion. *Aphasie und Diaschisis. Neurol. Centralbl.*, **Nov., 1906.** *Die Lokalisation im Gehirn*, **1914.**

Spiller, William G., studied lesion of the lenticular zone. *The Symptomatology of Lesions of the Lenticular Zone with some discussion of the Pathology of Aphasia. Journ. of Nervous and Ment. Dis.*, **1907.**

Moutier, a disciple of **Marie,‡** presented a good scheme of examining patients, in advance of anything then in use. He also published important statistics which showed that in 11 out of some 108 cases the lesion was not cortical but subcortical; in 8 the cortex of the left third frontal convolution was affected. All the other cases were almost entirely against Broca's theory of special area. At the most only 19 supported the theory. *L'Aphasie de Broca.* Paris, **1908.**

Collier, James, disagreed with the localisation theory. *Brain*, Vol. xxxi, **1908.**

Ballet. In a debate of the Société de Neurologie in 1908 he pointed out the essential difference between want of power to evoke words, and a true anarthria or articulatory defect, suggesting that motor aphasia was in reality apraxia.

Meyer, Adolph (1866-?) was an American pathologist and alienist born in Switzerland. He studied medicine in Paris, London, Edinburgh, Vienna, Berlin, and Zurich, then came to the United States where he lectured on neurology at the University of Chicago, 1892–1895, served as pathologist to the Illinois Eastern Hospital for the Insane at Kankakee, Ill., 1895–1902, and at Worcester, Mass., Insane Hospital, becoming professor of psychiatry at Cornell University Medical College in 1902, and at Johns Hopkins in 1910. He was president of the New York Psychiatric Society, and of the American Psychopathic Association. In his Harvey lecture he warned against the classic division of aphasia. He recognized that it was a functional problem, and devised an extensive test schema. He differentiated receptive disorders, intrinsic disorders, and disorders of emissive functions. Word deafness and disturbances of speech which he usually found to accompany it interested him particularly. The accompanying disturbances he termed "elaboration disorders," as for example, paraphrasia, defects in evoking

words and disturbances in reading and writing. His work is said to represent an advance over most of the views current in his day, and to be a transition step between the older doctrines of localization and the later studies of aphasia as complex and particular changes in mental functioning. *The Relation of the Auditory Center to Aphasia. Journ. f. Psychol. in Neur.*, **1908.**

Heilbronner, K., was a disciple of **Arnold Pick*** and after him it became common to use the term apraxia for defects of movement. *Die Aphäsischen Apraktischen und Agnostischen Störungen.* Berlin, **1910.**

Goldstein, Kurt, was born at Kattowitz, Germany, and became a neuropsychiatrist. He first assisted at the University Psychiatric Clinic in Breslau in 1903. He then became professor of neurology and psychiatry at the medical school of the University of Frankfurt, in 1914. He pointed out that cortical motor and sensory aphasia never occurred in pure form, but both were accompanied by disturbances of reading and writing, and that also, there is no isolated alexia or agraphia. He held that studies of the patient's response were futile unless the patient had exact knowledge of the situation from which it arose. He accordingly tried to make his examination a coherent whole, instead of a series of separate tests. *Ueber Aphasie. Beihefte z. Mediz Klinik*, **1910.**

Stauffenberg, von, affirmed mind-blindness to be a symptom which can never exist alone, but is associated with permanent or transitory difficulties of a higher order, such as those of memory, intelligence or speech, forming a syndrome. *Ueber Seelenblindheit. Arbeit a. d. Hirnanatom. Inst. in Zurich*, **1913.**

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FREQUENCY OF ARTICULATION SUBSTITUTIONS IN CHILDREN HANDICAPPED BY CEREBRAL PALSY

BERNEICE R. RUTHERFORD, M. S.,
Dowling School, Minneapolis

There seems to be a current opinion (1) that the defective speech among cerebral palsied children varies in type of sound substitution or omission according to the type of motor disturbance; (2) that the extrapyramidal group or those with athetoid movements have all speech sounds present but owing to the disturbed motor functioning these sounds vary from time to time, i. e., a given sound may be poor on one occasion and good on another, and therefore it is not to be classified as a substitution nor omission; and (3) that the "true spastic" or pyramidal group may have definite and constant speech sound substitutions or omissions.

This paper is written with a view to throwing some light upon this subject. The writer recognizes that the judgments concerning defective speech sounds and corrected speech sounds are quite subjective, and that standards for such judgment are different in different locations. However, these subjective judgments are presented for consideration.

For several years past charts have been kept on the speech of some hundred or more cases of cerebral palsy. Of these fifty-nine have been definitely differentially diagnosed by the orthopedic surgeon of the school. Frequently a sharp distinction is not possible because of the nature of the case. Whenever a case was diagnosed as "combined" or was in any way not clear cut it was not included in this study. Of the fifty-nine cases, thirty-two are of the athetoid or extrapyramidal group and twenty-seven are of the "true spastic" or pyramidal group. The differentiation observable in a physical way is in the writhing, bizarre movements of the extrapyramidal group and the more fixed and rigid position of the affected parts in the spastics. In this paper the athetoid group is to be considered as extrapyramidal in type, subcortical in origin and characterized by disordered motion. The spastic group is to be considered as pyramidal in type, cortical in origin and characterized by tight muscles and stiffness.

The procedure for testing for defective speech was the same in all cases. The Minneapolis word and sentence test was followed by conversations. Any speech sound was not listed as defective unless test and subsequent conversations revealed that the speech sound

was being continually omitted or had a constant substitution. All of the speech sounds listed as defective for any one child have appeared on more than one year's chart except in cases of permanent correction. Permanent correction was so designated only after a child was heard to use the specific sound correctly for a period of at least four weeks.

In this study the distribution of the 59 cases is:

Extrapyramidal (Athetoid) 32

Pyramidal (Spastic) 27

- Diplegias, 9
- R. Hemiplegias, 13
- L. Hemiplegias. 3
- Paraplegias, 2

In point of frequency of speech sounds for which a substitution is used, the following is the order:

Extrapyramidal
- s, z
- l
- sh, ch, j
- r
- θ, ð
- f, v
- c, k, g
- vowels
- h
- t, d

Spas. Diplegia
- s, z, sh, ch, j
- l
- r, vowels
- θ, ð
- f, v
- c, k, g
- h

Spas. R. Hemi.
- s, z
- sh, ch, j
- r, θ, ð
- l
- f, v, vowels

Spas. L. Hemi.
- s, z
- l, r, θ, ð, t, d

Paraplegias
- s, z, θ, ð
- l, sh, ch
- j, r, f, v
- c, k, g
- vowels
- h, t, d

In all instances "s" and "z" are the most frequently defective speech sounds; "l" sound seems to be second in frequency; then there seems to be some overlapping except in the Extrapyramidal group.

In the Extrapyramidal group "s" and "z" sounds hold first place with 29 cases or 94% of the 32 cases having continued difficulty with it; five were of the lateral emission type; "l" sound was found to be consistently defective in 28 cases or 87½% of them; "sh," "ch," and "j" sound in 24 cases or 75%; "r" sound in 17 cases or 54%; and

θ and ε in 16 cases or 50%. The other defective speech sounds are found scattered from 11 cases to 6 cases. They include in order of frequency f, v, c, k, g, vowels, h, t, d.

Among the pyramidal group, 18 cases or 67% of the 27 cases have defective "s" and "z," 2 were lateral emissions. Of these 4 were paraplegias, 2 were left hemiplegias, 6 were right hemiplegias, and 7 were diplegias. 12 cases or 40% have defective speech sound of sh, ch, j. Of these one was a paraplegia, 5 were right hemiplegias and 7 were diplegias. "l" sound, θ and ε sounds each have 11 cases or 34%. The diplegia group have more cases of multiple defective sounds than do any of the other pyramidal groups. Of the pyramidal and extrapyramidal groups, however, the latter have 69% (22 cases) having more than 3 defective speech sounds to 37% (10 cases) of the former.

Of the 32 extrapyramidal cases 6 had nasal speech; while of the 27 pyramidals 1 right hemiplegia and 1 paraplegia had nasal speech. Three of the extrapyramidals also stuttered, and 3 others had stuttered at some time or other; among the pyramidals 2 diplegias and 1 right hemiplegia also stuttered, while 1 right hemiplegia had stuttered at an earlier age.

It would seem from this study that (1) extrapyramidal speech defect cases do have very definite and persistent sound substitutions; (2) pyramidal cases also have definite and persistent speech sound substitutions; (3) the order of frequency of sound substitutions is practically the same in the extrapyramidal and pyramidal groups.

NEWER PRACTICES IN READING IN THE ELEMENTARY SCHOOL. By M. McBroom (Editorial Chairman). Washington: 17th Yearbook of the Department of Elementary School Principals of the National Education Association, Vol. 17, No. 7, July, 1938, pp. 704, $2.00.

This monograph, written by fifty-one different authors, represents the best information we have on how to teach reading in the elementary school, and how to prevent and correct reading disorders. Chapters of especial interest are: *Discovering Minor Reading Difficulties and Their Causes, Diagnosing Serious Reading Difficulties in the Primary Grades, Correcting the Reading Disabilities of Nine Selected Pupils, A Practical Approach to Remedial Reading, A Co-operative Project in Remedial Reading, The Art of Choral Speaking,* and *The Speech Aspects of Reading Readiness.* This last by Irene Poole Davis not only clarifies the relation of speech to reading, but also gives a great deal of definite information on language genetics with which every speech correctionist should be familiar. There are several bibliographies, in all, 245 references.

C. H. V.

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AMERICAN SPEECH CORRECTION ASSOCIATION

Speech Correction Section of the N. E. A.

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University of Oregon Medical School

AN ABSTRACT

CARE OF SPEECH HANDICAPS IN EUROPEAN COUNTRIES*

It is evident that the first attempts at general help for speech defective people were worked out in connection with schools for deaf-mutes. After medicine had gained a fuller knowledge of anatomy, physiology, pathology, neuro-psychiatry, and especially of brain pathology and voice physiology, successful attempts were made by the medical schools to solve the riddle of the causes, symptoms, and treatment of speech and voice disorders. It is interesting to observe that, with the increasing knowledge of speech defects, the question of professional training and prophylaxis became outstanding. The available statistics from countries where speech corrective work has been in progress over a period of years indicate that best results can be obtained through finding and treating the disorders early in childhood. Although European countries have led in the field of prophylaxis and treatment in speech corrective work, the project has not become a unified one in any country. It seems that there has been a lack of a general system to carry out the early diagnosis and treatment of speech disorders, but the development of speech corrective work is leading toward a program of early prophylaxis, without which all attempts in the field of speech corrective work seem insufficiently effective.

(Entire paper will appear in Proceedings of the N. E. A. for July, 1939.)

S. S. HAWK.

*From speech delivered by Dr. Margaret Ringer, University of Oregon Medical School, at Seventy-seventh Annual Convention of the National Education Association, Division of Speech Corrective Work, July 4, 1939.